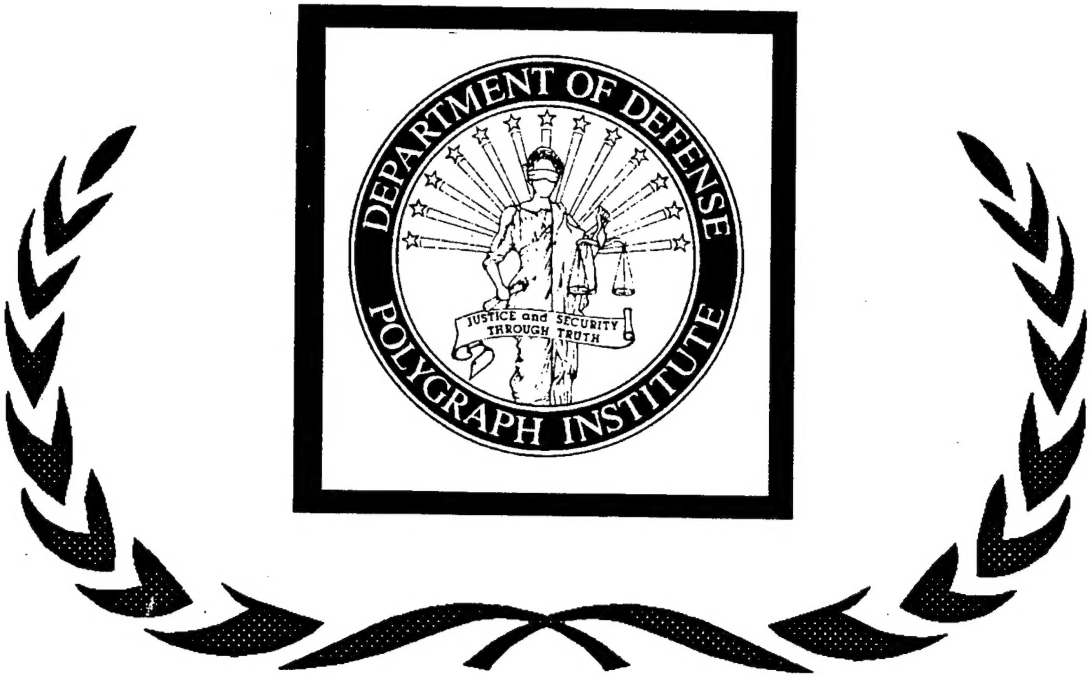

Cardiovascular Indices of Guilty Knowledge

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June 1994

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Director's Foreword

This study is one in a series of studies to explore a variety of sensors for recording cardiovascular physiological activities (CPA). The purpose of this line of research is to find a sensor for recording CPA that will: (1) be easier to quantify; (2) be more sensitive to basic CPA; (3) not cause discomfort to the examinee; and (4) allow lengthier test formats should circumstances require them. This study was designed to examine various non-invasive measures of cardiovascular activity, using impedance cardiography and blood pressure measures.

While there is some evidence here that subject veracity may be accurately ascertained using advanced technology and procedures, assumptions based on this study alone should be tempered with caution, because of the small subject population tested. While it is true that impedance cardiography is non-invasive, it is intrusive and may not be practical for the psychophysiological detection of deception (PDD) in the field. The demonstrated ability of technologically advanced instruments and processes to differentiate among multiple patterns of cardiovascular responses has been shown. This should encourage further investigations into the development of more sensitive and practical instruments and procedures for analyzing and categorizing these patterns for PDD.

Michael H. Capps
Director

Acknowledgments

I particularly appreciated the gracious assistance at the Department of Defense Polygraph Institute (DoDPI) of Drs. Gordon Barland and Barbara Carlton, MSgt Randy Reynolds, Brenda Smith, Fred Fisher, and the invited, constructive criticism of my experimental protocol, provided by Drs. Barland and Carlton. I dedicate this report to my father, Cecil R. Miller, Ph.D., who sparked my interest in psychophysiology during the late 1950's. This research was supported by the Department of Defense Polygraph Institute project DoDPI93-P-0012, under a grant administered by the Office of Naval Research, United States Navy (contract #N00014-93-C-0079). The views expressed in this article are those of the author and do not reflect the official policy or position of the Department of Defense or the U.S. Government.

Cardiovascular Indices of Guilty Knowledge
James C. Miller, Ph.D., C.P.E.

ABSTRACT

The project attempted to determine, through a multivariate analysis of cardiovascular function using impedance cardiography, what pattern of autonomic activity was reliably associated with concealed knowledge. The hypothesis was that (h_1) reliable, meaningful changes in the autonomic control of cardiovascular function are associated with concealed knowledge. The subjects were ten non-obese males with no prior polygraph experience. Four trials of six questions each were used. All trials were constructed like the Numbers Test (Furedy and Ben-Shakar, 1991). The first trial was based upon playing cards. The second through fourth trials involved the subject's year, month and day of birth, respectively. Digitized physiological data were acquired for 30 sec following each stimulus. The breathing pattern, skin conductance responses, electrocardiogram, impedance cardiogram and fingertip pulse pressure were recorded. The SCR-based detection rates for lies, 62.5% and 67.5%, were well above the expected random guessing rate of 20%. These relatively high rates suggested that different autonomic responses were associated with lies than with true responses. In turn, this observation suggested that differences in cardiovascular dynamics between lies and true responses may have been triggered by autonomic changes. The responses evoked in the cardiovascular measures by pre-Lie, lie, and post-Lie answers appeared to occur in the context of tonic activity in the cardiovascular components of the two autonomic branches with the highest level of coactivation preceding the pre-Lie truth and the lowest preceding the post-Lie truth. Most of the interesting phasic cardiodynamic activity following truthful and untruthful answers occurred in the first 10 or 11 beats following the subject's answer. The results showed that:

1. The pre-Lie condition was associated more than the Lie or post-Lie conditions with a tonic "fight-or-flight" response.
2. For the pre-Lie true response, TPR was highly reactive immediately following the stimulus for half of the subjects. For the other half, TPR was reactive several seconds later. For both groups, Q reacted inversely to TPR.
3. There were phasic cardiac indications (contractility, SV, IBI) of greater "relaxation," but a phasic, peripheral vascular indication (MAP) of greater "tension" for the Lie compared to the post-Lie response.

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Cardiovascular Indices of Guilty Knowledge

James C. Miller, Ph.D., C.P.E.

The forensic psychophysiologist works with many biophysical signals generated by the human body. Many of those signals are modified sharply by variable activities in a complex network of peripheral nerves called the autonomic nervous system (ANS). It appears that the presence of guilty knowledge may generate autonomic patterns which are differentiable from patterns associated with the absence of guilty knowledge [for example, "Dependent- and Independent-Variable Extensions of the Guilty Knowledge Test (GKT): Towards Field Applications," a symposium at the 32nd annual meeting of the Society for Psychophysiological Research, San Diego, CA, 15 Oct 1992].

The cardiovascular system is heavily influenced by the ANS. Thus, psychophysiological research has mined cardiovascular responses heavily, especially behaviorally induced heart rate (HR) and blood pressure (BP) changes. Unfortunately, the cardiovascular system is influenced by factors other than the ANS. Additionally, the easily measured parameters of cardiovascular function, such as HR and BP, do not accurately reflect autonomic influences. To understand the full context of changes in cardiovascular function in response to behavior, including the presence and absence of guilty knowledge, one must monitor the physical function of the heart.

The only practical, reliable, non-invasive tool available to the psychophysiologist to monitor the physical function of the heart is thoracic electrical impedance plethysmography, known also as impedance cardiography (ZCG; Miller and Horvath, 1978). This project was directed primarily at introducing the technique of impedance cardiography into forensic psychophysiology.

Activity in the two branches of the peripheral autonomic nervous system--the parasympathetic and (ortho)sympathetic branches--is tied loosely to behavior through an integrative process located presumably in the hypothalamus. The nature of the link is such that sympathetic activity may increase, and parasympathetic activity may decrease, with behavioral activation and cortical arousal. However, the degree of change in each branch is not wholly reciprocal, and change may occur in one branch without change in the other, or both branches may be active simultaneously. Additionally, one branch may predominate in the control of a function innervated by both branches. Finally, each of the two branches is segmented, and the segments within a branch may be differentially active.

Heart rate, due partly to the relative ease of acquisition of the electrocardiogram (ECG), has been the variable of choice in many investigations of arousal, activation, and stress during the last 25 years. Until the impedance cardiogram (ZCG) became available to psychophysiologists, heart rate in such investigations had not been viewed within the context of cardiovascular dynamics. However, heart rate may not be a controlled cardiovascular variable with respect to behavior. Instead, it may be a controlling variable that interacts with others to control blood pressure.

Heart rate is but one of two factors that determine cardiac output. The mathematical product of cardiac frequency (heart rate) and the volume of blood ejected per cycle (stroke volume) represents the flow rate (cardiac output) produced by the heart. Cardiac output, in turn, is responsive to the resistance to flow met in the body's vasculature, such that blood pressure (the mathematical product of flow and resistance) is maintained within a controlled range.

In the normal human, heart rate is responsive to signals that indicate changes in vascular system pressure and blood gas contents. These are the baro- and chemoreflexes, and they are relayed through autonomic pathways. Heart rate is also responsive to signals from the cortex and the limbic system, again relayed through autonomic pathways. Finally, heart rate is indirectly responsive to a biomechanical volume control that tends to stabilize cardiac output with respect to the flow of blood returning to the heart (Starling's Law of the Heart).

This brief discussion of cardiovascular function suggests that heart rate may not be the cardiovascular variable primarily controlled by the autonomic nervous system as behavior varies in the non-exercising individual. This being the case, the usefulness of heart rate as a single index of behavior in the psychophysiology or biofeedback laboratory has been called into question. The cardiac-somatic coupling hypothesis (Obrist *et al.*, 1970; Obrist, 1982) was instrumental in causing the psychophysiology research community to employ multivariate analysis techniques more widely in assessments of cardiovascular function.

Obrist argued that it was virtually impossible to comprehend the meaning of heart rate change, with respect to autonomic function, unless one considered heart rate in the context of whole-body and individual-organ metabolic or related (such as heat loss) demands. Impedance cardiography allows the non-invasive estimation of many of the whole-body cardiovascular parameters cited above. Its applicability to research using individuals who are not candidates for invasive examinations of cardiovascular dynamics appears obvious. However, as with any complex modern technological "advance," one must become intimately familiar with the strengths and weaknesses of the method to apply it with discrimination.

In this project, I attempted to determine, through a multivariate analysis of cardiovascular function, what pattern of autonomic activity was reliably associated with concealed knowledge. The theory was that reliable, meaningful changes in the autonomic control of cardiovascular function are associated with concealed knowledge. I also attempted to differentiate among four patterns of autonomic influence on cardiovascular function: reciprocal, co-active, sympathetic-dominant, and parasympathetic-dominant. The project consisted of a pilot study and a more formal investigation. Both are reported here.

PILOT STUDY METHODS

Subjects

The subjects were eleven non-obese males ranging in age from 20 to 31 years (mean 24.5 years). Seven were Caucasian and four were Afro-American. Eight had taken one or more polygraph tests previously and three had not. All were at least one hour post-prandial at the time of testing and none had consumed a high-fat meal within two hours of testing. The testing sessions began at 0730, 0930, 1130 or 1330. Each subject was paid for participating.

The data from the first subject were discarded due to technical deficiencies. The data from the second subject were discarded because the subject, a night shift worker, was unable to avoid drowsing during the session.

Procedures

The principal investigator explained the investigation to each subject, and each subject provided informed consent using a format approved by the Human Use Committee of the DOD Polygraph Institute, Ft. McClellan AL, where the data were collected. Each subject received at least one practice trial of the Numbers Test paradigm, and then was instrumented. Each subject completed at least five trials of the Numbers Test while instrumented.

The Numbers Test paradigm (Furedy and Ben-Shakhar, 1991) was arranged as follows. The subject selected and read one face-down playing card from the sixteen 2- through 5-value cards taken from a 52-card poker deck. The investigator was blind to the selection. The subject physically concealed the selected card in an envelope that he kept throughout the test. The information on that card, a number from two through five, was the subject of the subsequent questioning period.

The questioning period was divided into at least five trials. Within each trial, the first of the five questions asked was always "Is your number 1?" The data from this initial question were not analyzed; the question was present simply to begin each series in a standard manner. The subsequent four questions presented the numbers two through five in random order. All five questions were presented orally using the phrase, "Is your number n?" The timing of questions and the random order of the numbers two through five were provided to the investigator by a simple BASIC program running on one computer. A second computer was used to collect digitized physiological data and an event mark for each question within a trial. The interstimulus interval, between questions, was 30 sec. A trial was approximately three minutes long.

The subject answered "No" to each question asked. Thus, one of the subject's five responses within a trial was always a lie. The investigator attempted to identify the concealed number

through a real-time review of the physiological data. The intent of the protocol was to make the subject conceal information from the prying investigator.

For practice trials, numbers in the range 36 through 40 were used to avoid contamination of responses during the subsequent, instrumented trials. For the first three subjects, there were three practice trials and ten instrumented trials. This protocol proved too long. Thus, for the remaining eight subjects there was one practice trial and five instrumented trials. Also, the first three subjects practiced with the same numbers used for instrumented trials.

Instrumentation

Six channels of digitized physiological data, and one manual reading, were acquired for 20 sec following each stimulus. Each channel was digitized at 200 samples/sec. Each stimulus onset (the oral presentation of the stimulus number) was marked in a seventh digitized data channel.

The impedance cardiogram was recorded as two channels of data, dZ and dZ/dt, using the IFM/Minnesota Model 304B Impedance Cardiograph (Instrumentation for Medicine, Inc., Greenwich CT). The baseline impedance of the thorax, Z_0, was recorded manually. To acquire these signals, circumferential, aluminized mylar tape electrodes (IFM T-8001, Instrumentation for Medicine, Inc., Greenwich CT) were placed around the neck and abdomen (Miller and Horvath, 1978). The inter-electrode distance (L) was estimated as the mean of the measured front and back inter-electrode distances (Miller and Horvath, 1978). Calibration signals for dZ (0.1 ohm) and dZ/dt (1.0 ohm/sec) were recorded for each trial.

Fingertip pulse pressure was recorded using the Ohmeda Model 2300 Finapres BP Monitor (Ohmeda Division of the BOC Group Inc., Englewood CO), connected to a sensor placed on the middle phalanx of the second finger of the non-dominant hand. A 0-to-150 mmHg calibration was recorded at the beginning of each trial. The ECG was recorded using the SensorMedics R612 Dynograph and Type 9878 voltage coupler, with the "ECG" high pass filter, a 30 Hz low pass filter, and a 60-Hz notch filter (SensorMedics Corp., Anaheim CA). The ECG electrodes were attached approximately in the CR-5 locations (Simonson, 1971), with the LA electrode placed below the lower impedance cardiograph circumferential electrode.

The subject's breathing pattern was recorded using the Lafayette Factfinder polygraph and Model 76477-G signal conditioner (Lafayette Instrument Co., Lafayette IN), connected to a sensor that encircled the chest and responded to changes in chest circumference. Skin conductance level and responses (SCL, SCR) were recorded using the same polygraph and Model 76483-G GSG signal conditioner, connected to stainless steel electrodes attached to the middle phalanges of the first and third fingers of the non-dominant hand.

Data Reduction

The digitized data were collected using the AT-CODAS software (Release 5, Dataq Instruments, Inc., Akron OH) and 16-bit analog-to-digital conversion adapter in an 80386 IBM-clone personal computer. Each trial was recorded as a single CODAS binary data file. The data from the 20 seconds subsequent to the second through fifth number presentations within trials were extracted, using CODAS, as 7-channel ASCII files for further processing in spreadsheet software. Each of the 20 ASCII files (5 trials/subject x 4 stimuli/trial) extracted for further processing was approximately 350 kbytes, giving an overall ASCII database size of (20 files/subject x 10 subjects x 350 mbytes/file =) 70 mbytes. These files were compressed 79% and transported on 1.4-mbyte diskettes.

I selected the data of the last six subjects run during the pilot study for analysis. This was a manageable sample size in view of project time constraints, the need to develop a data reduction system, and the limited nature of the pilot study. For each of the six subjects, only the data from the first trial were examined. Within the first trial, two of the five responses were compared: the first true response and the lie response.

The two responses per subject selected presented a conservative view of the cardiovascular and SCR differences between lie and truth. Each true response preceded the paired lie response to a question. Thus, one would expect some autonomic lability during the true response. This is a common occurrence for true responses that precede the predicted occurrence of a lie. The lability during the true response was expected to reduce the effect size of cardiovascular and SCR differences observed between the true and lie responses.

The SCR data were scored in sequential context. Usually, a polygraph examiner will consider the sequential context of SCRs to a series of questions. For example, the largest SCR may occur following the lie, and large SCRs may accompany true responses preceding the lie. A novice (JCM) and an expert (GHB) examined the SCR data collected here.

For each question, the first six cardiac cycles were selected for analysis. This selection paralleled the data reduction approach of Furedy and Ben-Shakar (1991), who quantified the SCR during the 2nd through 4th seconds following stimulus presentation.

The 20-sec, ASCII file for each response, created by the CODAS software, was imported into a spreadsheet (Quattro Pro for Windows, Borland International Inc.). A template spreadsheet for the extraction of data from the 200-sample/sec file was also created. The template accepted data from the calibration files created by CODAS, as well as physiological data. Cell and cell range addresses were used as spreadsheet links to reduce the CODAS data to physiological units in the template. Spreadsheet XY plots were used to display calibration, ECG, dZ/dt, and SCL waveforms for visual pattern recognition.

Rapid processing of quantitative and graphic demands were supported by an Intel 80486/DX2 cpu operating at 66 mHz, with 32-bit communication to a video accelerator adapter. A 15-inch color monitor and full-screen graphic displays allowed ±1-sample visual identification of physiological data inflection points. The software was run in a Windows 3.1/DOS 6.2 operating environment (Microsoft Corp.) using 8 mbytes of RAM. Data were stored on a 340-mbyte hard disk drive.

For each response, I measured the SCL amplitude for the 2nd through 5th seconds following stimulus onset. The first cardiac cycle following the start of the 2nd second was labeled cycle 0. The subsequent six cardiac cycles were measured manually. The measures included:

Interbeat interval (IBI)
Stroke volume (SV)
Single-beat cardiac output (Q)
A correlate of contractility, the R-B interval (RB)
Mean arterial pressure (MAP)
Total peripheral resistance (TPR)

The ECG R wave was identified as the greatest positive slope of the ECG cycle. This approach reduced the uncertainty associated with sample-rate error in locating the peak of the R wave. The dZ/dt B wave (aortic valve opening) was identified as the last sample preceding the greatest positive slope in the cardiac dZ/dt cycle. The dZ/dt Z wave was identified as the greatest value in the cardiac dZ/dt cycle, and the dZ/dt X wave as the lowest value. Stroke volume and Q were calculated as in Miller and Horvath (1978). The contractility correlate (RB) was estimated as discussed by Siegel et al. (1969). The MAP was calculated as the point 2/3 through the range from diastolic to systolic pressure. Total peripheral resistance was calculated as the quotient of TPR and Q.

The template spreadsheets, one per subject, were linked to a summary spreadsheet. The latter spreadsheet was a resource for the extraction of data for statistical analyses and descriptive graphics.

The 20-sec strings of interbeat intervals were converted to regularly spaced samples and subjected to a discrete Fourier transform (code written some years ago by the investigator for small physiology projects) to determine the signal's energy content in the 0.1 to 0.4 Hz frequency band. This process provided some insight into cardiac-specific parasympathetic activity during lying.

PILOT STUDY RESULTS

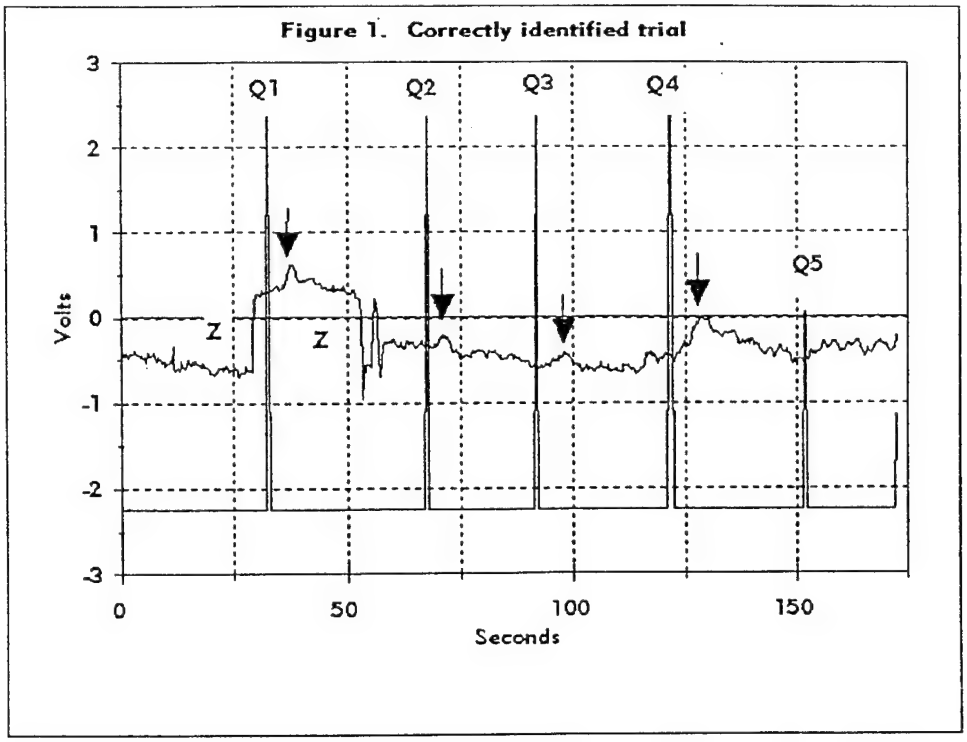
The manual data reduction process operated elegantly in allowing the paperless, accurate identification of measurement points (e.g., ECG R wave, dZ/dt B wave, etc.). This was the purpose of the design of the approach, and it met its goal. Visual inspection of the data revealed that the signal during the first true response from the last subject was unusable, so the second true response for that subject was used. Other than that instance, the quality of the data recorded was quite high.

Each 20-second data file expanded to about 1 mbyte in its spreadsheet version. The computer's speed and storage capacity easily supported the data reduction and analysis approach, as was intended.

The data reduction process suggested reasonable approaches to relatively simple pattern recognition software. The software would specifically address the calibration and scoring of SCL, ECG, dZ/dt, and pulse pressure tracings in a "lie detection" paradigm.

Skin Conductance Response

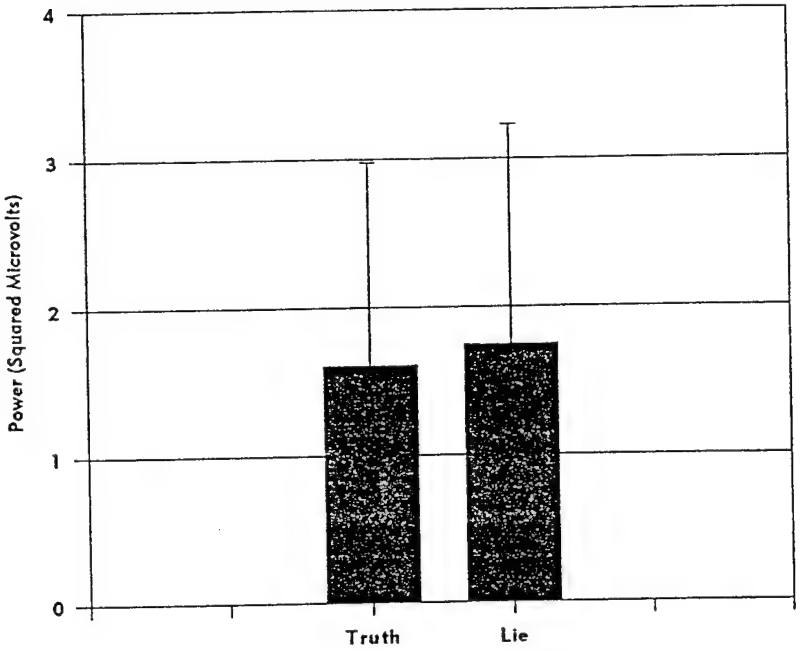
In three of the six cases, the subjects were so unresponsive that no classification was attempted. Both scorers correctly identified the lie on one out of the three remaining trials. Thus, their true positive detection rate was 0.33. This was slightly greater than the expected random detection rate of 0.25. They identified the same lie correctly, creating an inter-rater reliability of 1.0. The correctly identified trial is shown in Figure 1. The spikes labeled Q1 through Q5 mark the five questions asked of the subjects. The arrows mark the responses to the questions. The first response was always ignored in quantitative analyses. The fourth response was identified as the lie here because it was the last relatively large response. The responses to Q2 (truth) and Q4 (lie) were used for subsequent quantitative analyses. Similar approaches were taken to selecting responses in the other five trials.



Vagal Tone

Generally, the subjects produced bi-modal power spectra across the range, 0.05 to 1.0 Hz. One mode was in the 0.05 to 0.10 Hz range and one was in the 0.10 to 0.40 Hz range. Unfortunately, some sample epochs were slightly shorter than 20 sec, giving frequency bin widths slightly greater than 0.05 Hz. With that caveat in mind, I averaged the bins across subjects for the lie response and the true response. The total power in the 0.1 to 0.4 Hz range is summarized in Figure 2. Mean power (n = 6 subjects; error bar is +1 sd) was slightly greater during the 20 sec following the lie than it was following the true response. This pattern indicated that greater vagal tone was associated with the lie. The greater vagal tone should have produced a cardiac deceleration.

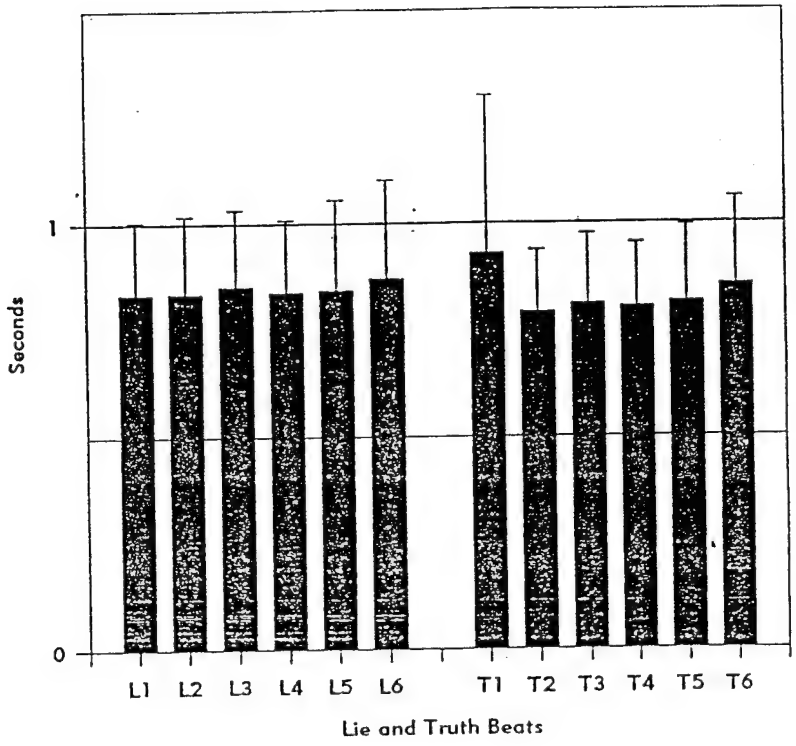
Figure 2. Mean vagal tone for truth and lie.



Cardiovascular Indices

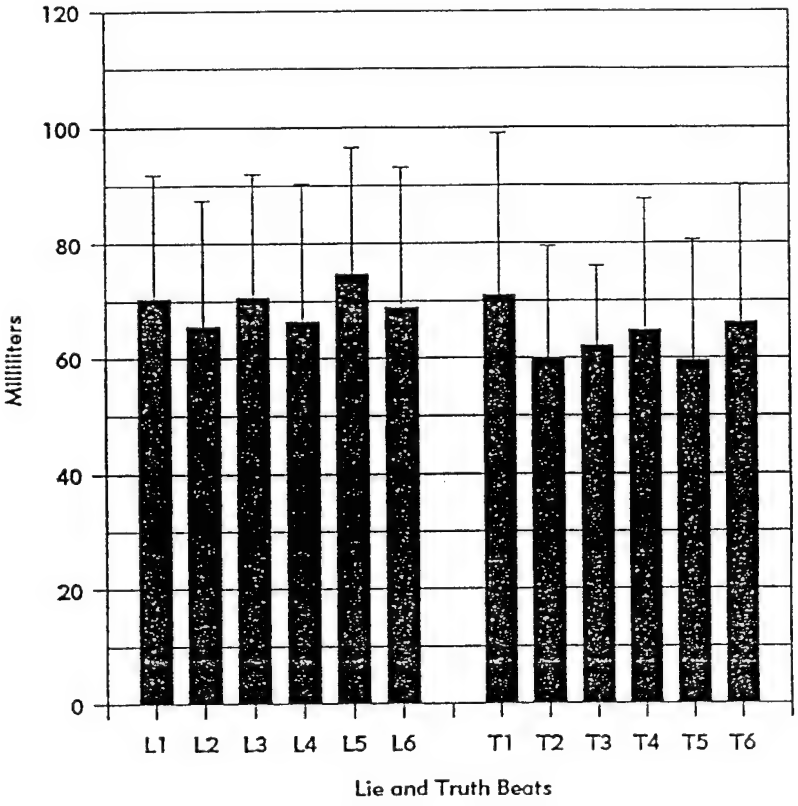
Consistent with the generally higher VT, measured IBI was slightly longer for lie responses than for true responses across beats 2 through 6 following the question (Figure 3; n = 6 subjects). Thus, one would expect increased SV due to a longer ventricular filling time across those beats (increased myocardial preload). One would also expect a mixed or canceling effect on calculated Q (the product of SV and HR) across those beats. The interbeat interval pattern of beats 1 through 6 for the true response also suggested a small cardiac acceleration from beat 1 to beat 2 that was absent for the lie response (Figure 3, also).

Figure 3. Interbeat interval.



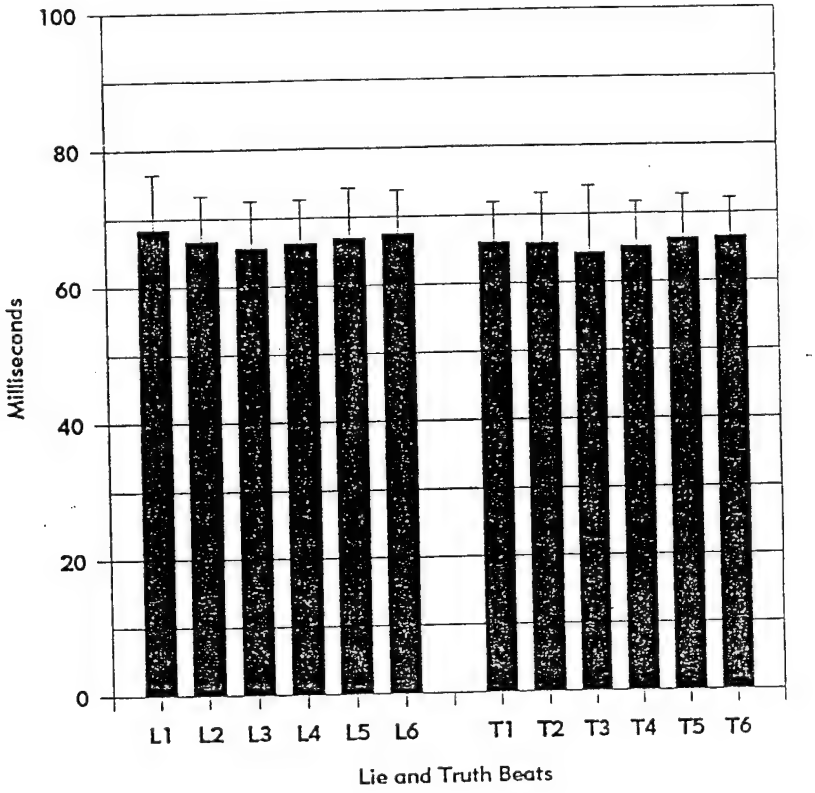
Consistent with the increased filling time, measured SV was slightly larger for lie responses than for true responses across beats 2 through 6 following the question (Figure 4; n = 6 subjects). The larger SV associated with the lie was also consistent with the increased myocardial contractility and decreased MAP associated with the lie, described below.

Figure 4. Stroke volume.



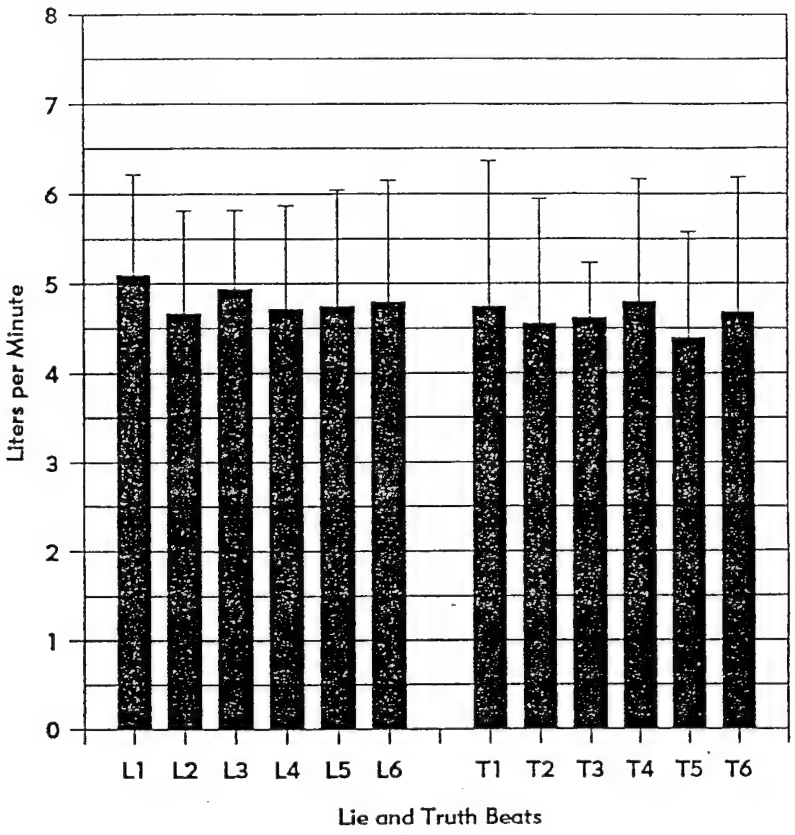
Myocardial contractility (measured as the R-B interval) was slightly higher (shorter R-B interval) for lie responses than for true responses across beats 1 through 6 following the question (Figure 5; n = 6). Thus, I expected and observed an increase in stroke volume, mediated by elevated sympathetic activity for those beats. The sympathetic effect would have been transmitted through beta-1 receptors and would have suppressed the negative inotropic effects of elevated parasympathetic activity by "accentuated antagonism" (Levy, 1977).

Figure 5. Contractility (R-B interval).



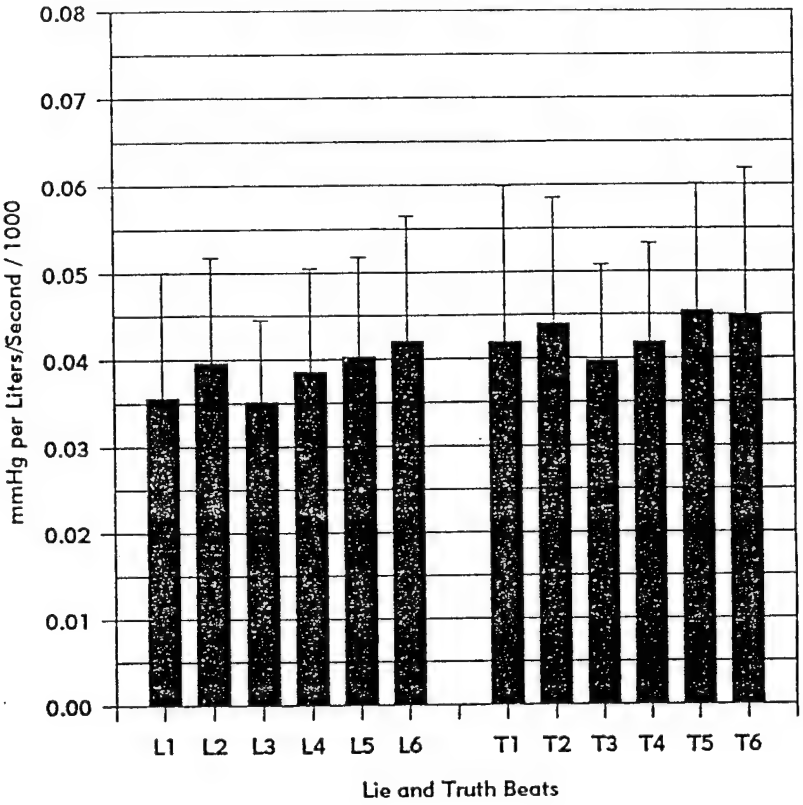
Conversely, I assumed that the observed elevation of vagal activity must have dominated elevated sympathetic beta-1 activity at the sino-atrial (SA) node to create the longer interbeat interval associated with the lie. These observations and assumptions suggest that the lies triggered coactivation of the sympathetic and parasympathetic control mechanisms for the cardiovascular system.

Figure 6. Cardiac output



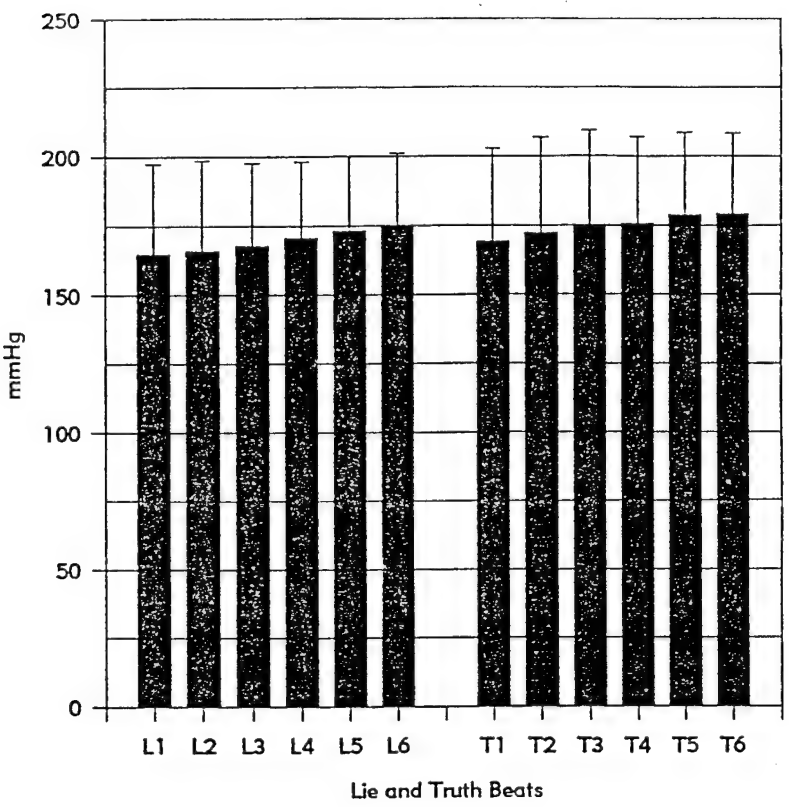
As expected, calculated Q was mixed across beats 1 through 6 when compared for the lie and true responses (Figure 6; n = 6). Calculated TPR was slightly lower for lie responses than for true responses across beats 1 through 6 following the question (Figure 7; n = 5 subjects). This observation is consistent with an elevated level of parasympathetic activity. It would also be consistent with an elevation of sympathetic beta-2 activity causing muscle vasodilation. It would not be consistent with an elevation of sympathetic alpha activity causing widespread vasoconstriction.

Figure 7. Total peripheral resistance.



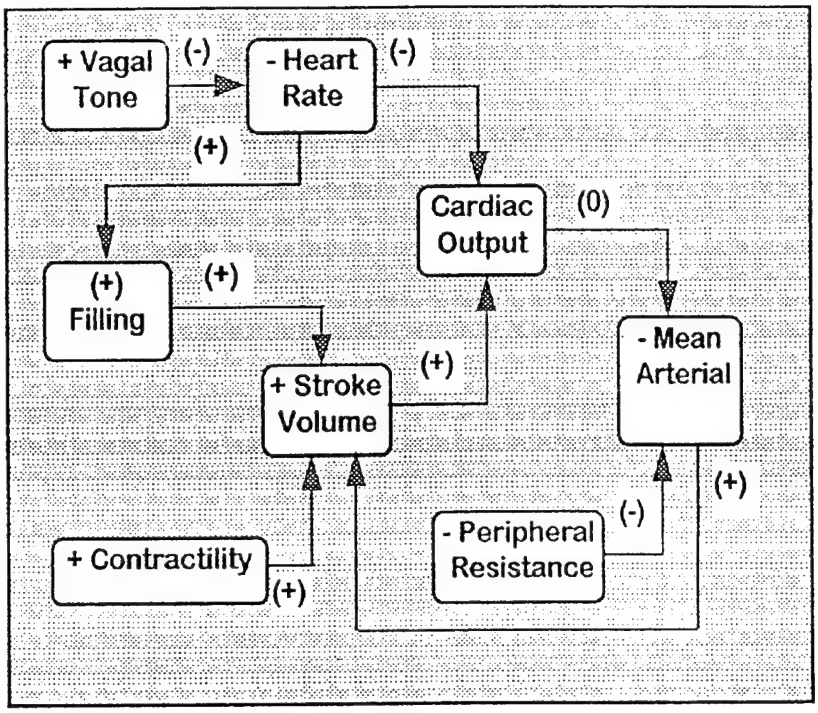
Mean arterial pressure is regulated, in large part, as the quotient of cardiac output and total peripheral resistance. Our measurements showed that MAP was slightly lower for the lie response than for the true response across beats 1 through 6 following the question (Figure 8; n = 5 subjects). The slightly lower MAP would have created a slightly lower myocardial afterload, consistent with the observed elevation in stroke volume.

Figure 8. Mean arterial pressure.



The apparent interactions of vagal tone and the other cardiovascular variables are depicted in the flow chart in Figure 9. The flow chart is summarized in the Conclusions section, below. In Figure 9, signs (+, -, 0) without parentheses represent observations. Parentheses represent assumptions.

Figure 9. Tentative model of cardiovascular response to lying.



PILOT STUDY CONCLUSIONS

One function of a pilot study may be to explore a speculation, producing testable hypotheses. That being the case, this pilot study was quite successful. Even though the "gold standard" of lie detection, the SCR, provided marginal information due to the lack focus by the subjects and the conservative approach to the selection of data for analysis, the associated cardiovascular data told a consistent, meaningful story. That story provided testable hypotheses.

To summarize, I speculated on the basis of the pilot data that the following pattern of coactivation underlies the cardiovascular responses associated with lying:

1. Increased vagal tone acts through cholinergic receptors at the SA node, dominating sympathetic beta-1 activity, to produce a negative chronotropic effect on the heart (longer IBI, faster HR).

    a) The longer IBI provides a longer ventricular filling time and increased myocardial preload, and the slower heart rate should reduce Q.

2. Increased sympathetic beta-1 activity produces a positive inotropic effect on the heart (increased myocardial contractility).

3. SV is elevated by the increased myocardial preload and by the increased contractility.

4. The combined effects of slower HR and increased SV cancel each other, producing no lie-specific effects on Q.

5. Increased parasympathetic activity and increased sympathetic beta-2 activity trigger peripheral vasodilation and a reduction in TPR. Reduced sympathetic alpha activity could, in theory, have a vasodilatory effect as well, but the genesis of reduced alpha activity in a coactivation pattern is difficult to discern.

6. The effect of the reduced TPR, working in concert with the unchanged cardiac output, reduces MAP.

    a) The reduced MAP feeds back to provide reduced myocardial afterload, enhancing SV.

For the investigation, then, we were provided with the following testable hypotheses: lying will be associated with increased vagal tone, IBI, contractility, and SV, and with decreased TPR and MAP. Lying may also suppress cardiac acceleration in response to a question.

INVESTIGATION METHODS

Experimental Design

Overall, the experiment was correlational in nature, using the type of response (lie, truth) as the categorical criterion variable and using dependent, continuous physiological measures distributed along a lie-truth continuum. The design was similar to a single-factor, 3-level analysis of variance (ANOVA) with type of answer as the factor and the answer conditions, pre-Lie, lie, and post-Lie, as the three levels. The pre- and post-Lie responses were true responses. The hypotheses to be tested included the following:

1. Compared to the pre- and post responses, the lie response will present significantly greater VT, IBI, myocardial contractility, and SV, and significantly lower TPR and MAP.
2. Compared to the pre- and post responses, the lie response will produce a significant suppression of cardiac acceleration in response to a question.
3. One or more cardiovascular measures will predict the occurrence of a lie at a statistically higher rate than chance.
4. One or more cardiovascular measures combined with the SCR measure will predict the occurrence of a lie at a statistically higher rate than SCR alone.

Data analyses were limited to empirical, visual assessments of group mean data presented in graphs. Large degrees of inter-subject variability diminished the value of estimating statistical reliability for this particular analysis. Patterns in the group mean data were assumed to represent general, fundamental physiological responses that were small compared to relatively random physiological system noise.

Subjects

The subjects were ten non-obese males ranging in age from 19 to 25 (mean 21.8 years). Half were Caucasian and half were Afro-American. They were paid $30.00 for participating. Informed consent was obtained as before.

Procedure

The foremost concern for the design of the experimental procedure was to increase the likelihood, above that of the pilot study, that the subjects would focus on the behavior associated with lying. The design of the pilot study and its resulting SCR data suggested a lack of focus on the part of the subjects. Thus, I introduced four fundamental changes to the experimental procedure.

First, I included personal data as topics for questioning. Specifically, the year, month and date of birth were three of the four topics addressed. Second, the subject was turned 90 degrees to sit face-to-face with the investigator. This confrontational seating arrangement is not used in field polygraph examinations.

Third, the subject was offered a $5.00 bonus for each birthdate question (year, month and day) for which I failed to identify the lie. This carried the potential of being a 50% bonus for participating as a subject. Finally, the question topics and answers were heterogeneous across trials, reducing the likelihood that the subject would orient to a question-answer pair in one trial based upon the structure of a previous trial.

Four trials of six questions each were used. All trials were constructed, again, like the Numbers Test (Furedy and Ben-Shakar, 1991). The first trial, again, was based upon playing cards. However, the card selection was made from the twenty 2- through 6-valued cards. The BASIC program used for question timing and randomization was modified to handle these and other changes to the procedure. As in Phase 1, the first question and its physiological responses were not used in the data set. However, there was one more question per trial in the experimental procedure (six questions, total) than in the pilot study (five questions, total).

The second through fourth trials involved the subject's year, month and day of birth, respectively. After the card test, the subject wrote his birth year, month and day on a card, keeping the investigator blind to the information. The year, month and date possibilities were narrowed to five choices each with the following procedure. Five-year periods (for example, 1970-74) were listed sequentially with 1-year lags for the period that included all possible birth years for the age range allowed for the subjects (18 to 35). Each lag was printed on a new line on a single sheet of paper. Thus, the subject's year of birth appeared on five lines of the page. The subject selected any one of the five 5-year periods that contained his birth year. Similar pages and selections were used for the birth month and day. Sample pages are appended (Appendix A). These selections were used interactively with the BASIC program during the experimental procedure to create 6-question trials.

The interstimulus interval was 36 seconds, of which 30 seconds were dedicated to data collection and the other six to system adjustment, if required, and the orally presented text of the question (for example, "Were you born in ...?"). No practice trials were given. Subjects were run at 0900, 1100 or 1400 hours. The subject received feedback about the success or failure of his deception after the card trial and at the end of the third birthdate trial. The feedback was based solely upon the subject's SCRs.

Instrumentation

I reduced the number of channels of physiological data from six to five. The presence of the Z_0 channel was unnecessary for scoring purposes, and data file sizes were large enough without extra data.

Data Reduction

The data from the 30 seconds subsequent to the second through sixth questions in each trial were extracted as 6-channel ASCII files. The longer data epochs (30 seconds) were designed to allow 30-sec assessments of vagal tone. Each of the 20 ASCII files (4 trials/subject x 5 questions/trial) extracted for spreadsheet processing was approximately 550 kbytes, giving an overall ASCII database size of (20 files/subject x 10 subjects x 550 kbytes/file =) 1100 mbytes (1.1 gbytes).

On about 60% of the trials, the lie occurred in response to question 3, 4 or 5 (Appendix B). Within these trials, at least one pre-Lie true response and at least one post-Lie true response were available for analysis. The distribution of these trials with the lie in position 3 through 5 was spread across subjects such that two subjects experienced one occurrence, four experienced two occurrences, and four experienced three occurrences. The data selected for analysis came from these trials, one trial per subject. When more than one trial per subject was available, the trial with the most correctly interpreted SCR pattern was selected.

Measures were extracted from three responses within each selected trial (pre-Lie truth, Lie and post-Lie truth). The pre- and post-Lie responses used were those immediately preceding and following the lie. Two measures were extracted from each response as time series: skin conductance (SCR) and the interbeat interval (IBI). The IBI series were transformed to the frequency domain and spectral power in the .1 to .4 Hz band was summed as an index of vagal tone. Six measures were extracted from the dZ/dt and Finapres wave forms, and with reference to the ECG: IBI (and heart rate, HR), RBI, SV, Q, MAP, and TPR. They were compared across the three levels, pre-Lie truth, lie, and post-Lie truth (pre, lie, post).

The ECG channel of each selected trial's ASCII raw data file was processed to detect the maximum upward slope of the R wave, using a BASIC program, rwav.bas[1]. The sample numbers of those maximum slopes, representing the times of occurrences of R waves, were saved in ASCII files (*.rwv). The sample number files were processed by another program, ibisam.bas, to transform the R wave occurrences into a two-sample-per-second time series (Porges and Bohrer, 1990) and save the time series data (*.tsr). The

[1] This and the five data processing programs mentioned subsequently, all written in Microsoft Extended Basic, version 7.1, were written by and are available from the investigator.

0th beat for each time series occurred during the presentation of the stimulus. Each series was 25 seconds long (50 samples).

In another program, vtmdft.bas, the raw time series data for each subject were zero suppressed, 10% (5) of the scores on each end of the time series were tapered to zero using a half-cosine function (Bingham et al. 1967 ref.), and the 25-sec, 50-score time series was transformed to the frequency domain with a discrete Fourier transform. The transform produced a power spectrum with a fundamental frequency and bin width of 0.04 Hz.

To reduce the unwanted effects of between-subject differences in lability and total power, the raw power scores for the frequency bins were standardized (z-scores) within subjects. Power observations in all bins for one subject, across the three responses within the trial, were used to compute the mean and standard deviation for that subject's standard scores. The z-scores of the raw power values were averaged across subjects and reviewed graphically. The z-scores for the bins, $0.12 < x \leq 0.40$ Hz, were summed to represent vagal tone (Porges and Bohrer, 1990).

Calibration data were extracted from the CODAS ASCII files by the program, calsumm.bas, and saved (*.cal). The calibration data were combined with the R wave location data by the program, zcg.bas. This program found the dZ/dt B, Z, and X inflection points and the FinaPres low and high pressures, converted to physiological values, and stored IBI, contractility, SV, Q, MAP, and TPR data (*.zcg). Finally, the program, prep.bas, organized the ZCG data by variable for spreadsheet input.

INVESTIGATION RESULTS

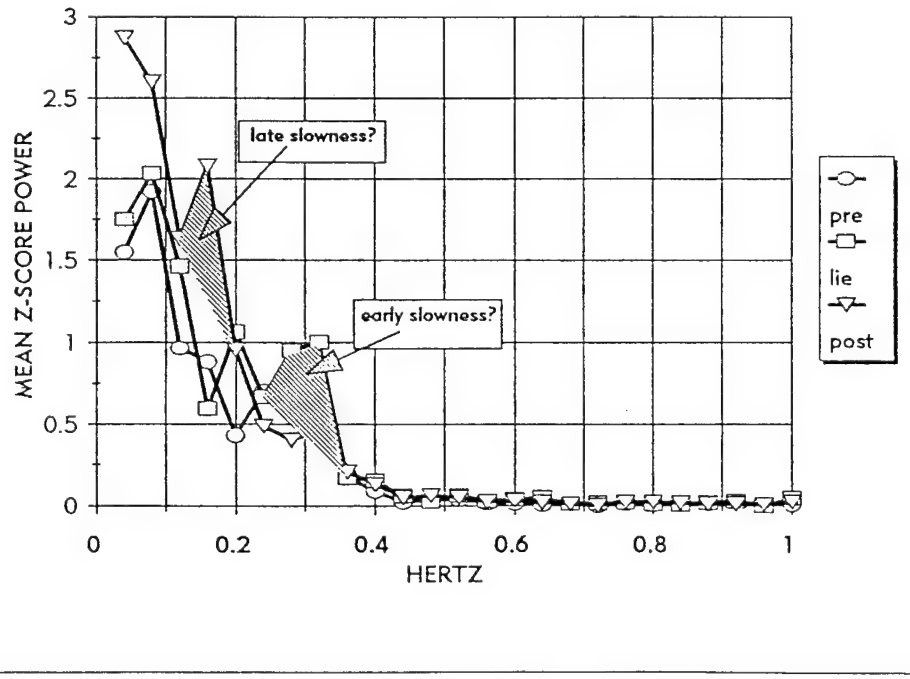
Skin Conductance Response

The distribution of correctly identified lies for the novice (62.5%) and the experienced (67.5%) scorers is tabulated in Appendix C. The useable trials were as shown in Appendix B. For seven of the subjects, trials in which both scorers correctly identified the lie were available for cardiovascular analyses. For two subjects (S2 and S3), the only trials available were trials in which only one scorer correctly identified the lie. For one subject (S5), the only trial available was one in which neither scorer correctly identified the lie. Of the ten trials selected, two were card trials, three were year trials, three were month trials, and two were day trials. The SCR responses are shown and described in Appendix D.

Vagal Tone

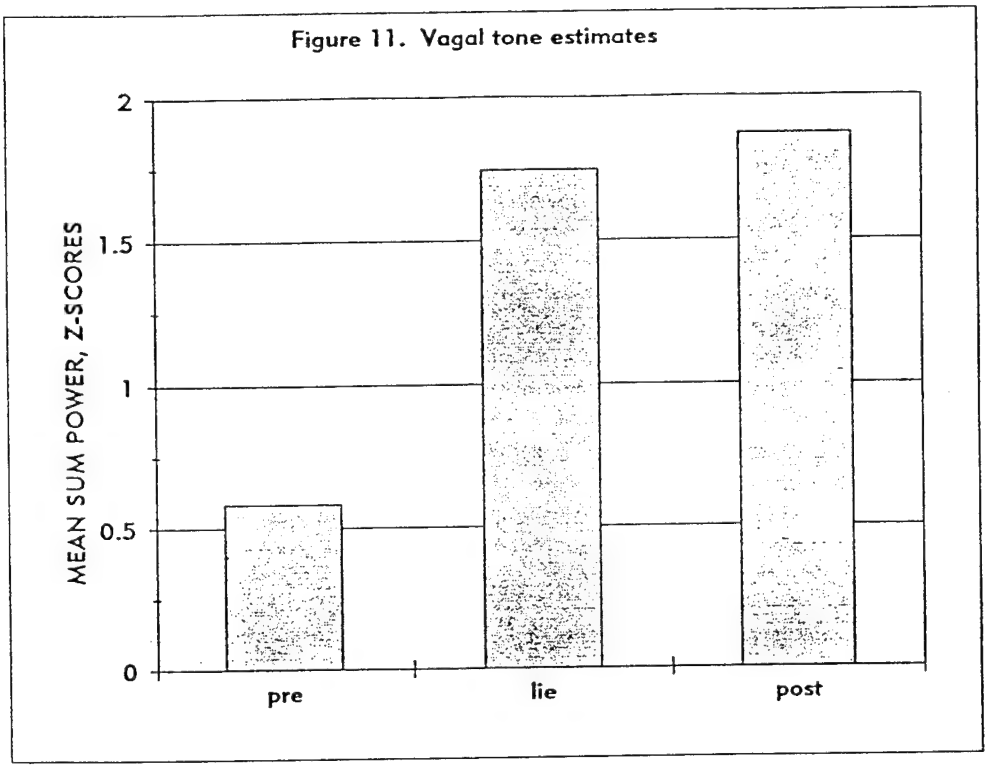
The mean z-score power spectra for the three responses are shown in Figure 10. A relative high for the Lie occurred in the 0.28 through 0.32 Hz range. This pattern may have been associated with ventilation rate and/or with the mean increase in IBI observed early in the lie epochs (see below). A relative high for the post-Lie truth (post) occurred in the 0.16 Hz bin. This pattern may have been associated

Figure 10. IBI power spectra



with the mean increase in IBI observed late in the post-Lie epochs (see below). A plot of the logarithms of these points revealed no differences among responses at frequencies above 0.40 Hz.

Figure 11. Vagal tone estimates



The mean z-score VT estimates for the three responses are shown in Figure 11. Using the group mean, VT appeared to be higher during the Lie and during the post-Lie truth than during the pre-Lie truth (pre). However, the VT estimates were actually rank ordered within subjects as shown in Table 1, with 1 being the highest VT and 3 the lowest level of VT. Thus, the mode (4 subjects) of the rank-order distribution placed the Lie VT *between* the two truths. The lie VT was highest in three subjects and lowest in two subjects compared to the truth VT values.

Table 1. Rank orders of Vagal Tone estimates.

Rank Order			No. of Subjects
Pre	Lie	Post	
3	2	1	4
2	1	3	3
2	3	1	1
1	3	2	1
1	2	3	1

Interbeat Interval

The beat-to-beat patterns of IBI using raw score and within-subject z-score data are shown in Figures 12 and 13, respectively. During the first seven beats, IBI increased (HR decrease of about 6 bt/min) for the Lie response and was elevated above the IBIs (about 2 bt/min) for the two true responses. There was an early, very small biphasic pattern for the two true responses, with its nadir at beat 4 (about 2 bt/min) and its peak at beat 7 (about 5-6 bt/min). Late in the epoch, IBI for the pre-Lie truth was reduced (HR increase of about 2 bt/min).

Contractility

The beat-to-beat patterns of the R-B interval using raw score and within-subject z-score data are shown in Figures 14 and 15, respectively. Contractility appeared to be relatively low (longer RB interval) for the pre-Lie truth during the first five beats, with a reversal upward at beat 3.

Figure 12. Interbeat interval

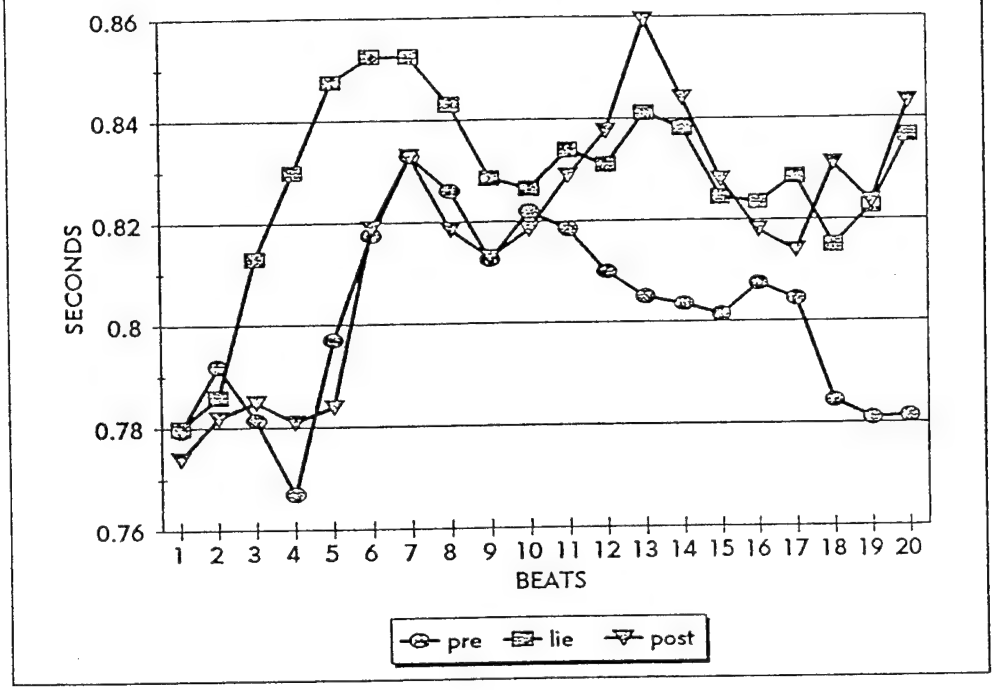


Figure 13. Interbeat interval

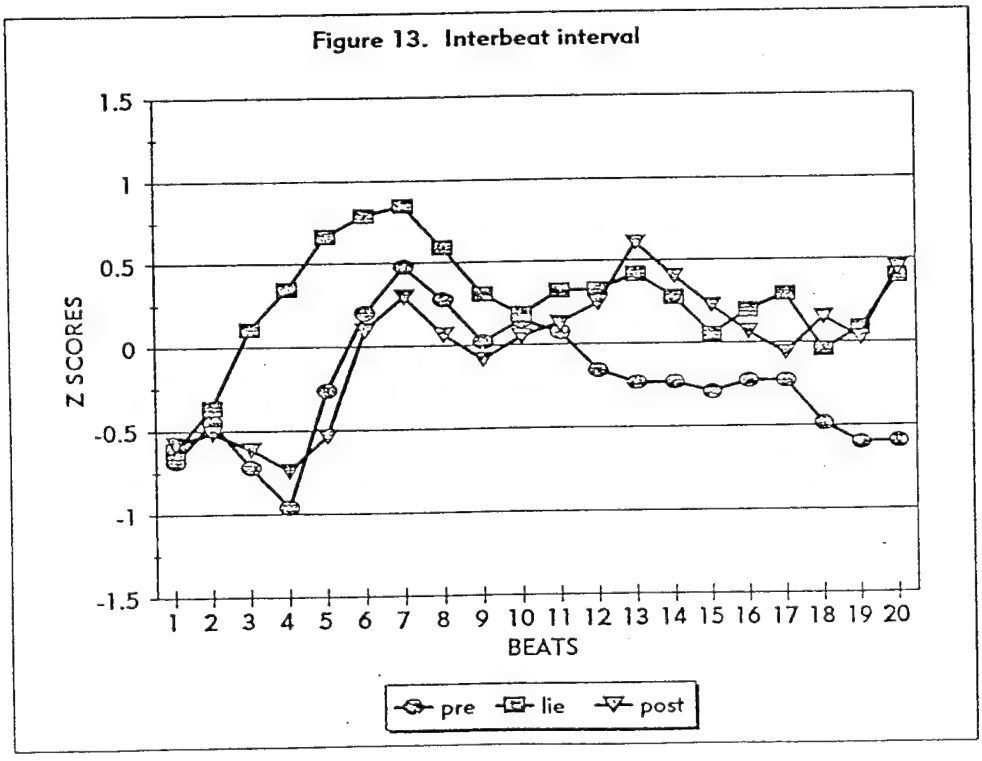


Figure 14. Contractility (RB)

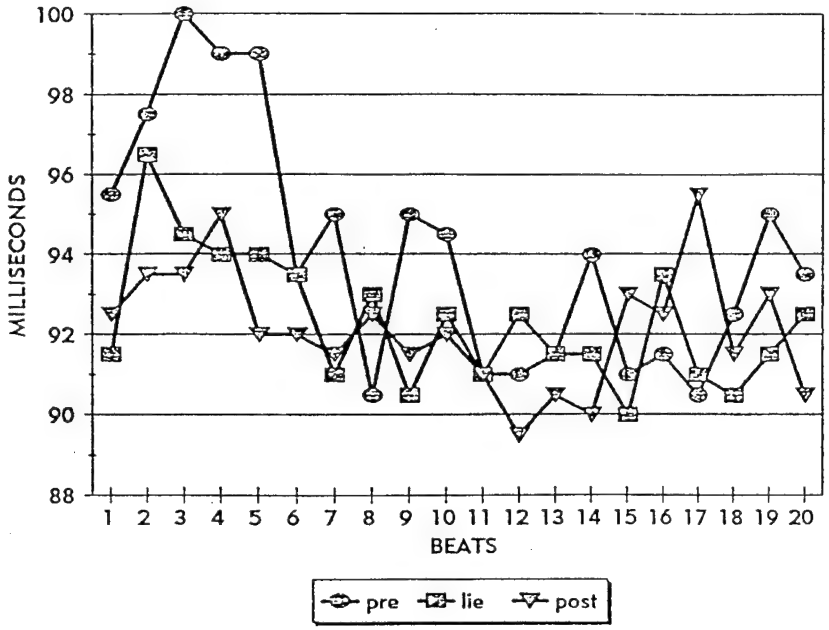
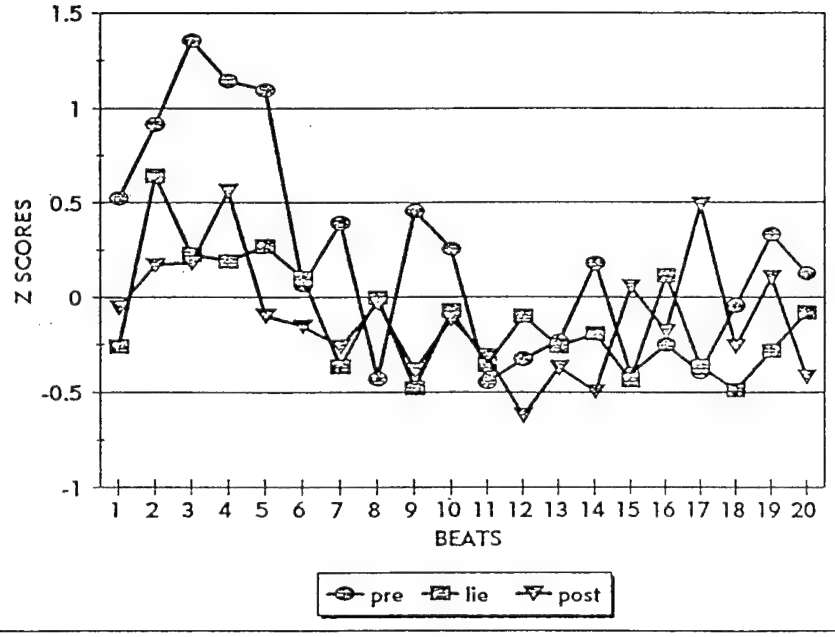


Figure 15. Contractility (RB)



Stroke Volume

The beat-to-beat patterns of SV using raw score and within-subject z-score data are shown in Figures 16 and 17, respectively. There was an early, biphasic response for all three responses with its nadir at beat 2. During beats 1 through 5, the z-score responses were rank-ordered, from highest to lowest: post-Lie, Lie, pre-Lie.

Cardiac Output

The beat-to-beat patterns of Q using raw score and within-subject z-score data are shown in Figures 18 and 19, respectively. Like SV, there was an early, biphasic response for all three responses with its nadir at beat 2. Like SV, but during beats 1 through 4, the z-score responses were rank-ordered, from highest to lowest: post-Lie, Lie, pre-Lie.

Figure 16. Stroke volume

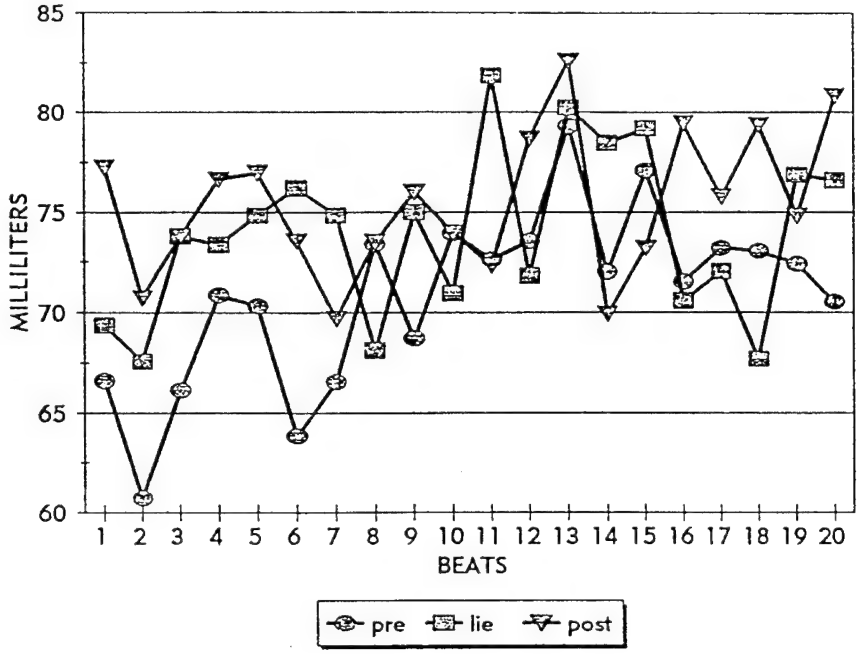


Figure 17. Stroke volume

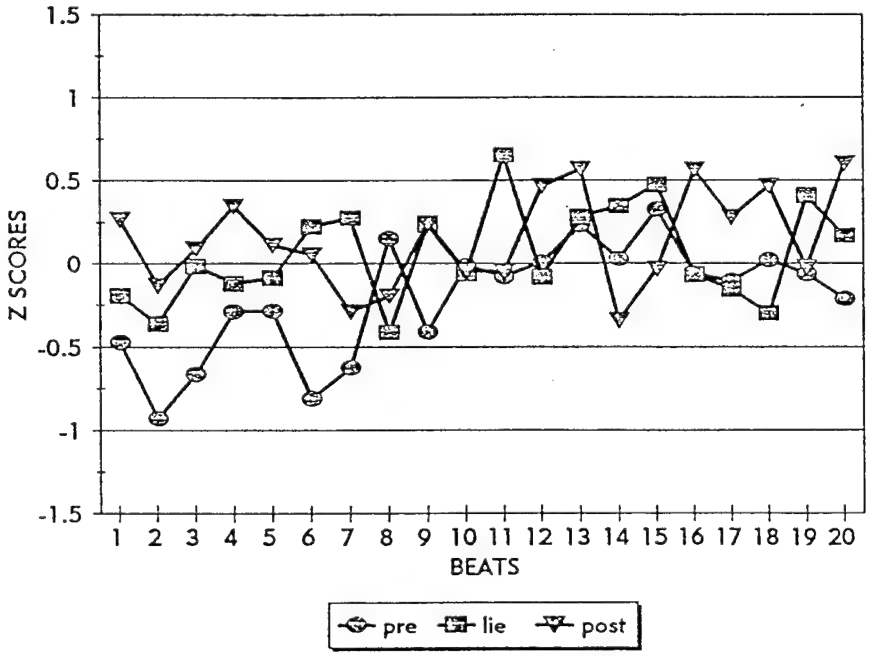


Figure 18. Cardiac output

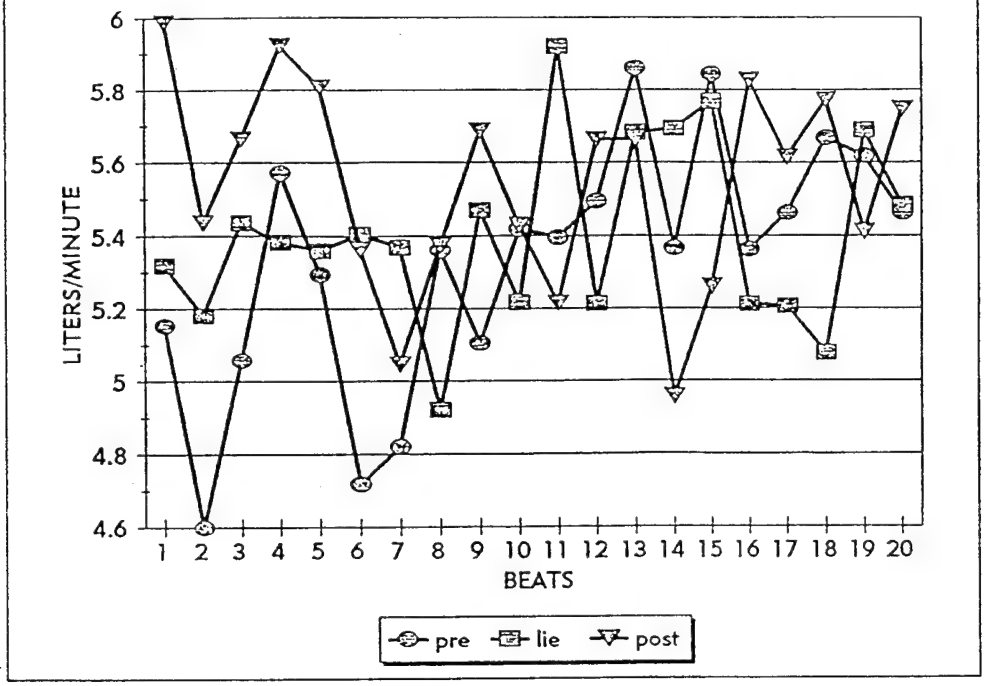
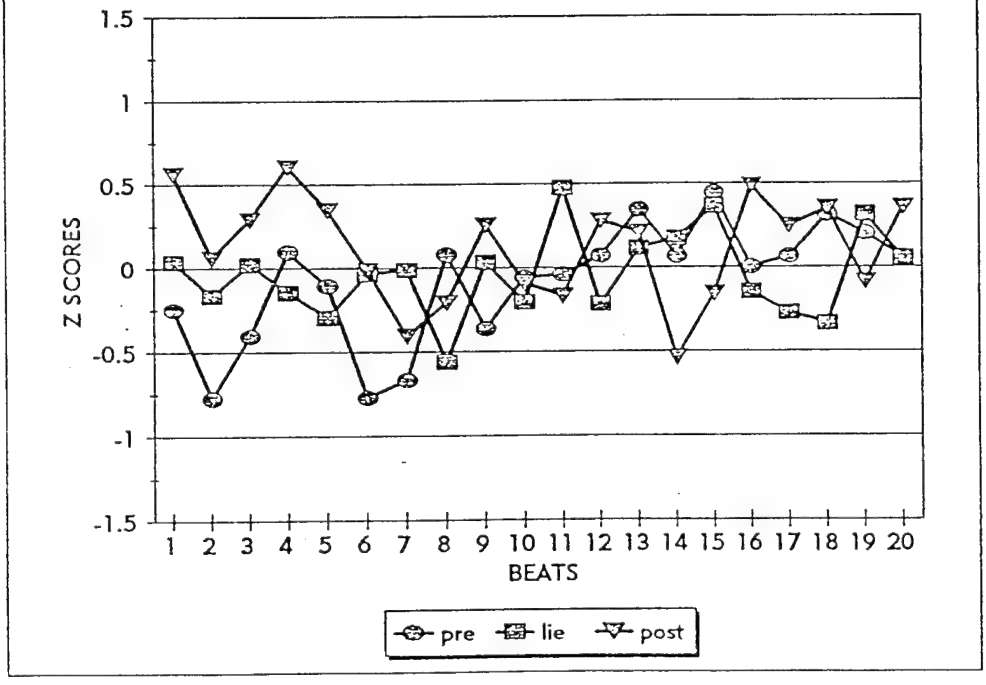


Figure 19. Cardiac output



Total Peripheral Resistance

The beat-to-beat patterns of TPR using raw score and within-subject z-score data are shown in Figures 20 and 21, respectively. There was an early, small biphasic response for all three responses with its peak at beat 2. During beats 1 through 3, the z-score responses were rank-ordered, from highest to lowest: pre-Lie, Lie, post-Lie. The early pattern of TPR was the reciprocal of the early patterns for SV and Q.

Mean Arterial Pressure

The beat-to-beat patterns of MAP using raw score and within-subject z-score data are shown in Figures 22 and 23, respectively. For the post-Lie truth, MAP increased through beat 6, then decreased. For the Lie, MAP started higher and increased through beat 4 then decreased. For the pre-Lie truth, MAP started the highest and increased through beat 3, showed a slight downward change, increased further through beat 6, then decreased. During beats 1 through 6, the z-score responses were generally rank-ordered, from highest to lowest: pre-Lie, Lie, post-Lie.

Figure 20. Total peripheral resist.

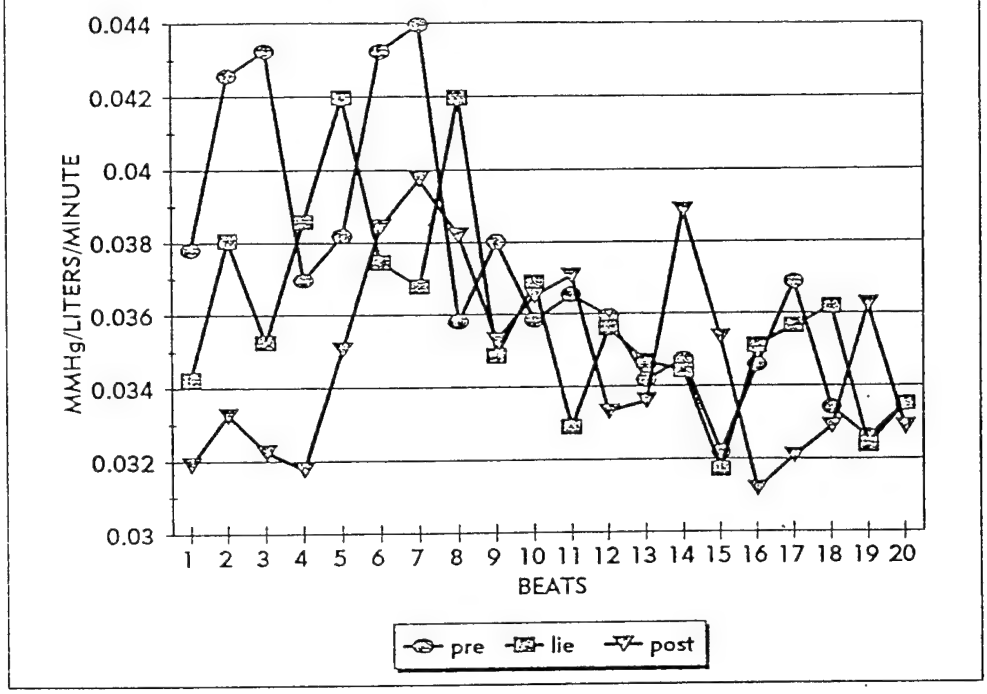


Figure 21. Total peripheral resist.

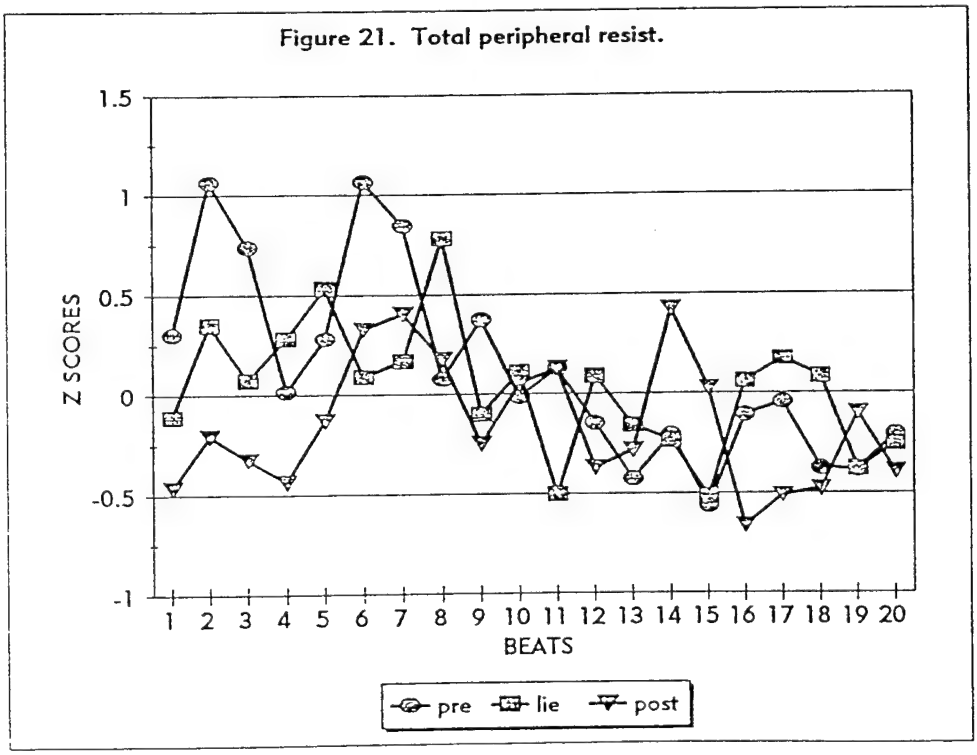


Figure 22. Mean arterial pressure

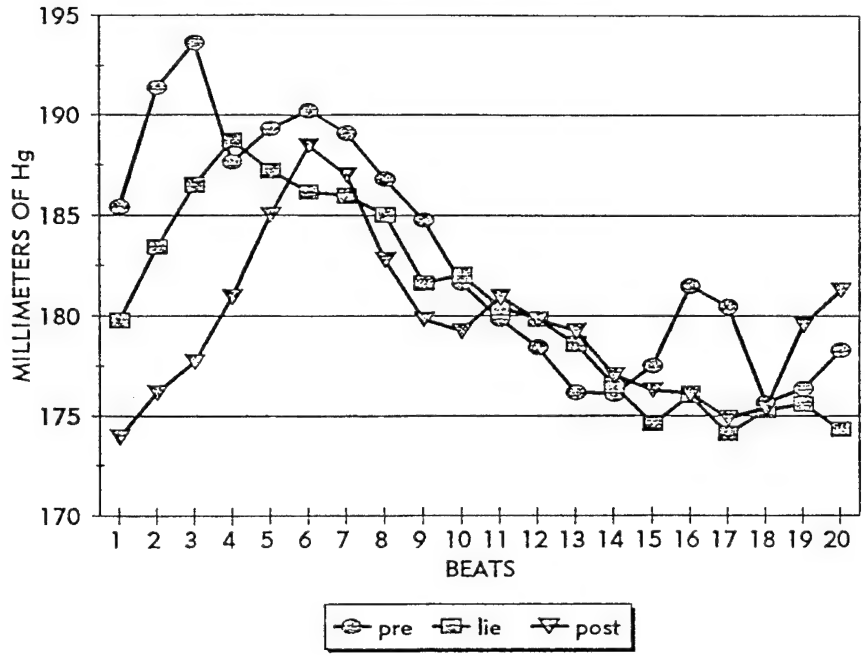
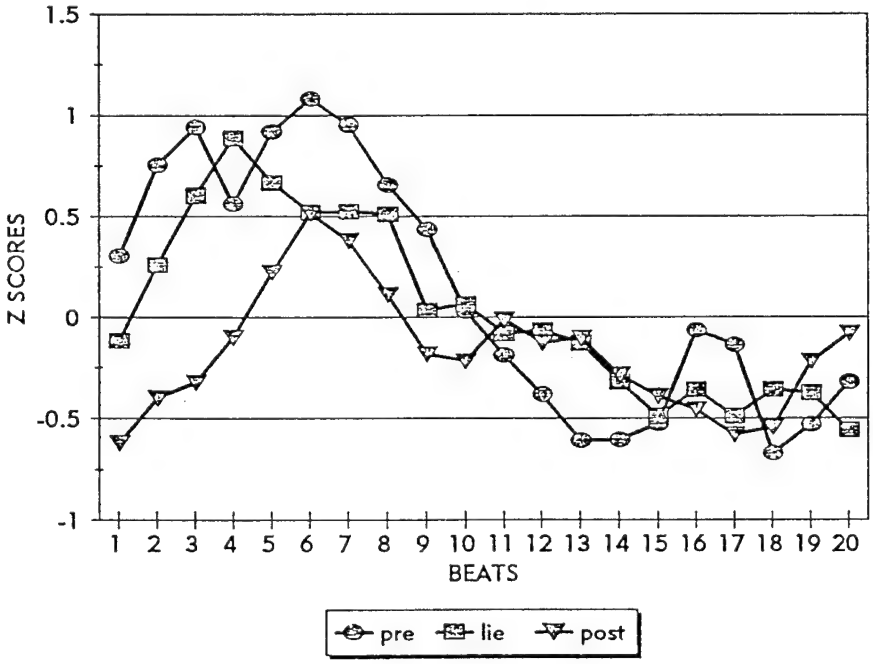


Figure 23. Mean arterial pressure

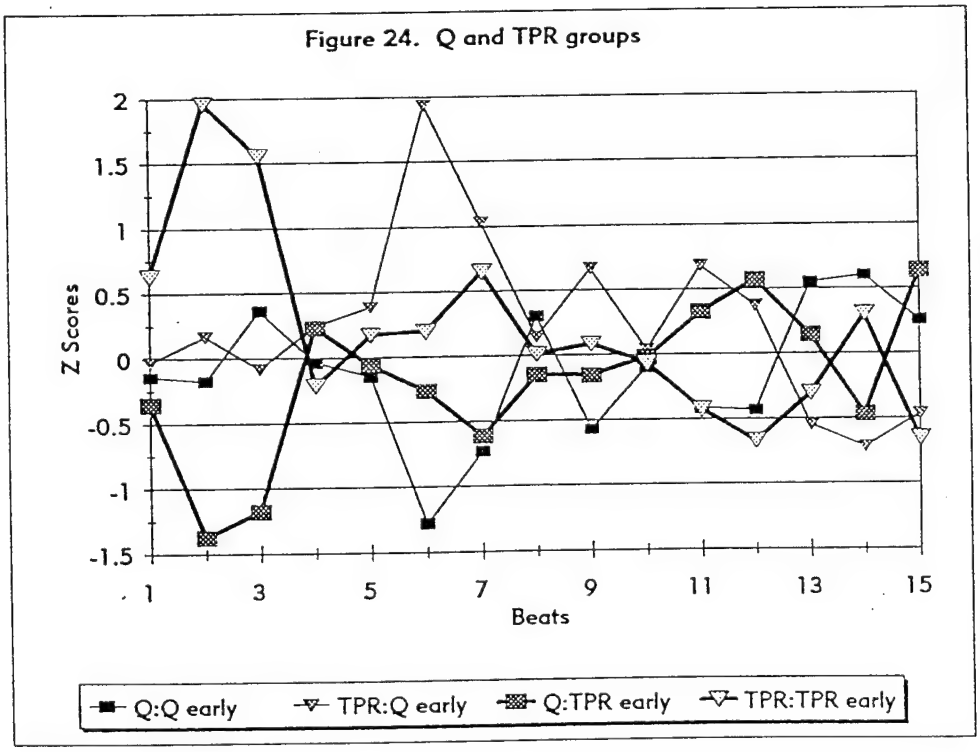


Individual Differences

Subjects were sorted into two categories of reactivity: an early Q response after the question and answer, or an early TPR response. The sort was based upon individual z-score values for the two variables for beats 2 through 11 in the pre-Lie condition. An early Q response was characterized by greater-than-group-mean Q z-score values, without similarly high values for TPR, during the first several beats after the stimulus. An early TPR response was characterized by greater-than-group-mean TPR z-score values, without similarly high values for Q, during the first several beats after the stimulus. There were 5 subjects in each group. Their mean Q and TPR values are shown in Figure 24. The early Q responders (thin lines) actually showed little reaction until beat 6, when TPR (triangles) was elevated and Q (squares) was depressed. The early TPR responders (thick lines) showed a strong TPR response across beats 2 and 3, with a concurrent depression of Q.

The two patterns of Q responding explained the apparent transient increase of Q across beats 2 through 6 shown in Figures 18 and 19. In fact, some subjects produced a Q nadir at beat 2 and some at beat 6. The inverse was true for the apparent transient depression of TPR across beats 2 through 6 shown in Figures 20 and 21. A review of the distribution of correctly identified lies (Appendix C) and the patterns of SCL across each individual's session (Appendix D) revealed no obvious connection between individual cardiovascular response differences and individual SCL patterns.

Figure 24. Q and TPR groups



Score Variabilities

Across the six z-score plots (odd-numbered Figures from 13 through 23), it was informative to know the amount of variability represented on each plot. The coefficients of variability for the plots (grand sd ÷ grand mean) were as shown in Table 2, in rank order. They sorted into two groups, around 17-20% (TPR, SV, and Q) and around 5-6% (RB, IBI, and MAP).

Table 2. Grand standard deviations (Std Dev) and coefficients of variability (CV) for the cardiovascular measures.

Measure	Std Dev	CV
Total peripheral resistance (TPR)	0.0071 mmHg/L/min	20%
Stroke volume (SV)	13 ml	17%
Cardiac output (Q)	0.95 L/min	17%
Contractility (RB interval)	5.4 msec	6%
Interbeat interval (IBI)	0.052 sec	6%
Mean arterial pressure (MAP)	9.9 mmHg	5%

DISCUSSION

The SCR-based detection rates for lies, 62.5% and 67.5%, were well above the expected random guessing rate of 20%. These relatively high rates suggested that different autonomic responses were associated with lies than with true responses. In turn, this observation suggested that differences in cardiovascular dynamics between lies and true responses may have been triggered by autonomic changes.

Vagal Tone

The VT data were equivocal. The mode of the rank-order distribution placed the Lie VT *between* the two truths. The lie VT was highest in three subjects and lowest in two subjects. The VT data did *not* support the hypothesis of increased vagal tone associated with lying. This finding may be due to measurement error or to the underlying psychophysiology. Perhaps the measurement technique should have been enhanced by removing non-linear trends from the IBI time series and by estimating coherence with breathing rate (Porges and Bohrer, 1990).

As discussed below, most of the interesting cardiodynamic activity following truthful and untruthful answers occurred in the first 10 or 11 beats in the epoch. The VT estimates came from 25 beats, and the influence of the first and last few beats of each epoch were minimized by the tapering process prior to the Fourier transform. Thus, very little of the evoked activity in each condition was reflected in the VT estimate. The technique should be modified so that the ten beats following the answer fall in the middle of the epoch used to estimate VT.

Tonic Patterns

The initial values of Q and SV were ranked similarly across the three conditions: post-Lie, Lie, pre-Lie (from highest to lowest, respectively). The rank order of the initial values of MAP and TPR was clearly reciprocal to Q and SV. From these observations, one may infer that:

1. Tonically high sympathetic alpha activity and/or
2. Tonically low sympathetic beta-2 activity and/or
3. Tonically low parasympathetic activity

caused greater peripheral vasoconstriction in the pre-Lie condition than in the post-Lie condition, with the Lie condition in between. The recovery of heart rate toward a higher level toward the end of the pre-Lie epoch was consistent with:

1. Tonically high sympathetic beta-1 activity and/or
2. Tonically low parasympathetic activity

during the pre-Lie period. The low initial value of contractility for the pre-Lie condition suggested tonically low sympathetic beta-1 activity. By a process of elimination, then, the tonic pattern may have been

1. Tonically high sympathetic alpha activity and/or
2. Tonically low sympathetic beta-2 activity and/or
3. Tonically low parasympathetic activity.

It appeared that the pre-Lie condition may have been associated more than the other two conditions with this particular tonic pattern.

Phasic Patterns

For the purposes of comparison and discussion, the post-Lie truth response patterns in the cardiovascular data serve as the "normal" response patterns for this experimental paradigm. Once the subject had told the lie, the SCR patterns suggested that he relaxed to some degree. The post-Lie truth response patterns were assumed to reflect this relatively relaxed state. They may also have resembled to some degree cardiovascular responses to questions in more relaxed settings. The discussion relies on the standard (z) score plots in the Results section.

Post-Lie Truth Patterns

The SV and Q patterns showed a 1-beat drop from a slightly elevated initial value, a small peak at beat 4, a decline, then great variability. The 1-beat drop would have been insignificant in the overall amount of variability except for its repeatability across the three conditions depicted in the plots. It would appear that the initial 1-beat drop in SV and Q was due to the rise in MAP and resulting myocardial afterload, and to the slight decrease in myocardial contractility. Filling time and resulting myocardial preload increased, but only slightly, for beat 2. Myocardial afterload was probably exacerbated for one beat by the positive intra-thoracic pressure associated with speaking the answer to the question posed by the investigator.

The MAP started at a low initial value, increased steadily through beat 6, then declined, driven primarily by TPR. Perhaps a:

1. Slowly decreasing tonic sympathetic alpha activity and/or a
2. Slowly increasing tonic sympathetic beta-2 activity and/or a
3. Slowly increasing tonic parasympathetic activity

was (were) reversed briefly during the post-Lie true response epoch. The slight (about 10 mmHg) increase in MAP was the most striking cardiodynamic change associated with a post-Lie, true response.

After beat 4, HR declined about 4 bt/min to a stable level and contractility returned to a stable level after a transient decrease. Perhaps these patterns reflected an increasing tonic parasympathetic activity following the Lie epoch.

Lie Patterns

I hypothesized that, compared to true responses,

1. The lie response would produce a significant suppression of cardiac acceleration in response to a question. In fact, the slight cardiac acceleration during beats 1-4 for the two kinds of truths was suppressed during the Lie. Instead of reaching a nadir at beat 4, the Lie IBI increased steadily from beat 1 and peaked at the same point as for the post-Lie truth, beat 7. Then IBI declined to an intermediate value.

2. The IBI would be higher with the Lie. In fact, IBI was greater during beats 1 through 9.

3. Contractility would be higher with the Lie. In fact, it was close to the post-Lie values for beats 1 through 6.

4. The SV would be higher with the Lie. In fact, it fell between the two true responses for beats 1 through 5.

5. The TPR and MAP would be lower with the Lie. In fact, it fell between the two true responses for beats 1 through 3.

These hypotheses were based upon a pilot paradigm that apparently elicited little emotional involvement from the subjects and which did not reveal different sympathetic-to-parasympathetic lag times between the Lie and the true response. The primary difference between the hypotheses and the experimental findings were for TPR and MAP. The pilot study results apparently replicated the pre-Lie to Lie differences in TPR and MAP noted here: a reduction from pre-Lie to Lie. Thus, the pilot study generated the wrong hypotheses that these values would be lower for a lie than for a true response.

The early slopes of several of the measures were of interest when compared to the post-Lie response:

1. The Lie contractility reached a nadir at beat 2 instead of beat 4;
2. The Lie SV presented the slight 1-beat drop, then rose slightly through beat 7 instead of beat 4, and
3. The Lie IBI increased earlier, though with lesser slope. However,
4. The Lie MAP increased steadily through beat 4 instead of beat 6.

Thus, there were cardiac indications (contractility, SV, IBI) of greater "relaxation," but a peripheral vascular indication of greater "tension." The slight rise in MAP was the most striking feature of the *post*-Lie epoch. But with the Lie, MAP rose even more sharply and peaked earlier. These observations suggested that, compared to the post-Lie condition, the Lie may have been characterized by:

1. Weaker sympathetic beta-1 activity and/or
2. Stronger tonic sympathetic beta-2 activity and/or
3. Stronger tonic parasympathetic activity

Pre-Lie Truth Patterns

The early patterns of IBI were similar across the three conditions. However, the pre-Lie truth contractility presented a much more pronounced trough from beats 1 through 6 than for the Lie or the post-Lie truth, suggesting a suppression of sympathetic function during that period. (Contractility did not show individual differences related to Q and TPR)

Stroke volume, rather than HR, appeared to dominate in the control of Q across the three conditions. The patterns in Figures 16-19 were quite similar. Both Q and MAP phasic patterns seemed to be dominated by TPR.

For half of the subjects, the TPR was highly reactive immediately (beats 2-3) following the stimulus. For the other half, TPR was reactive later, around beat 6. For both groups, Q reacted inversely to TPR. The implication of the difference in TPR reactivity for detecting concealed information was obscure. Perhaps the early-TPR group was among the sub-population that responds to stress with passive coping and concurrent elevated TPR rather than elevated Q (e.g., Sherwood et al., 1990).

Overview

The three conditions, then, were characterized primarily as follows:

1. The rank orders of the initial values of MAP, TPR, Q, and SV, suggested that the pre-Lie condition was associated more than the other two conditions with a tonic "fight-or-flight" response.
2. For the pre-Lie response, TPR was highly reactive immediately following the stimulus for half of the subjects. For the other half, TPR was reactive several seconds later. For both groups, Q reacted inversely to TPR.
3. There were phasic cardiac indications (contractility, SV, IBI) of greater "relaxation," but a phasic, peripheral vascular indication (MAP) of greater "tension" for the Lie compared to the post-Lie response.

Whether these observations are robust enough to provide a lie detection capability remains to be seen. To answer that question, at least several investigations will be

kinds of concealed information. Second the findings should be replicated using other kinds of concealed information. Third, investigations of the individual differences in TPR responding should be pursued. Finally, tests are required of the hypotheses that:

1. One or more cardiovascular measures will predict the occurrence of a lie at a statistically higher rate than chance, and
2. One or more cardiovascular measures combined with the SCR measure will predict the occurrence of a lie at a statistically higher rate than SCR alone.

If the pattern suggested here of tonic and evoked responses is consistent across experimental replications, an optimal linear model of the cardiodynamics of lying may be generated through discriminant analyses using data from this investigation and replications. The discriminant functions may then be examined for their detection values in validation studies.

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Appendix A. Selection-narrowing forms for birth year, month and date.

5-Year Categories

_____ 1956-1957-1958-1959-1960
_____ 1957-1958-1959-1960-1961
_____ 1958-1959-1960-1961-1962
_____ 1959-1960-1961-1962-1963
_____ 1960-1961-1962-1963-1964
_____ 1961-1962-1963-1964-1965
_____ 1962-1963-1964-1965-1966
_____ 1963-1964-1965-1966-1967
_____ 1964-1965-1966-1967-1968
_____ 1965-1966-1967-1968-1969
_____ 1966-1967-1968-1969-1970
_____ 1967-1968-1969-1970-1971
_____ 1968-1969-1970-1971-1972
_____ 1969-1970-1971-1972-1973
_____ 1970-1971-1972-1973-1974
_____ 1971-1972-1973-1974-1975
_____ 1972-1973-1974-1975-1976
_____ 1973-1974-1975-1976-1977
_____ 1974-1975-1976-1977-1978
_____ 1975-1976-1977-1978-1979

5-Month Categories

_____ Jan-Feb-Mar-Apr-May
_____ Feb-Mar-Apr-May-Jun
_____ Mar-Apr-May-Jun-Jul
_____ Apr-May-Jun-Jul-Aug
_____ May-Jun-Jul-Aug-Sep
_____ Jun-Jul-Aug-Sep-Oct
_____ Jul-Aug-Sep-Oct-Nov
_____ Aug-Sep-Oct-Nov-Dec
_____ Sep-Oct-Nov-Dec-Jan
_____ Oct-Nov-Dec-Jan-Feb
_____ Nov-Dec-Jan-Feb-Mar
_____ Dec-Jan-Feb-Mar-Apr

5-Day Categories

_____ 1-2-3-4-5
_____ 2-3-4-5-6
_____ 3-4-5-6-7
_____ 4-5-6-7-8
_____ 5-6-7-8-9
_____ 6-7-8-9-10
_____ 7-8-9-10-11
_____ 8-9-10-11-12
_____ 9-10-11-12-13
_____ 10-11-12-13-14
_____ 11-12-13-14-15
_____ 12-13-14-15-16
_____ 13-14-15-16-17
_____ 14-15-16-17-18
_____ 15-16-17-18-19
_____ 16-17-18-19-20
_____ 17-18-19-20-21
_____ 18-19-20-21-22
_____ 19-20-21-22-23
_____ 20-21-22-23-24
_____ 21-22-23-24-25
_____ 22-23-24-25-26
_____ 23-24-25-26-27
_____ 24-25-26-27-28
_____ 25-26-27-28-29
_____ 26-27-28-29-30
_____ 27-28-29-30-31
_____ 28-29-30-31-1
_____ 29-30-31-1-2
_____ 30-31-1-2-3
_____ 31-1-2-3-4

Appendix B. Distribution of usable SCR recordings for the experimental procedure.

Subject	Cards	Year	Month	Day
1	2 or 6	2 C *	2 or 6	2 C
2	0 C	1 C *	2 or 6	0 C
3	0 C	2 or 6	1 C	1 C *
4	2 or 6	1 C	2 C *	0 C
5	0 C *	0 C	2 or 6	0 C
6	2 or 6	2 or 6	2 or 6	2 C *
7	2 C *	2 or 6	2 or 6	2 or 6
8	1 C	2 or 6	2 C *	2 or 6
9	2 or 6	2 C	2 C *	2 C
10	2 or 6	2 C *	2 or 6	1 C

Where:

- "2 or 6" means the lie was at the ends of the series, in position 2 or 6;
- "0 C," "1 C" and "2 C" mean 0, 1 or 2 scorers correctly identified the lie, respectively; and

* shows the trials selected for analysis.

Appendix C. The distribution of correctly identified lies for the novice and the experienced scorers (based solely on SCR) for the experimental procedure.

	Card		Year		Month		Day		Tot	
Subj	N	E	N	E	N	E	N	E	N	E
1	1	1	1	1	1	1	1	1	4	4
2	0	0	1	0	0	0	0	0	1	0
3	0	0	0	0	0	1	0	1	0	2
4	1	1	0	1	1	1	0	0	2	3
5	0	0	0	0	1	1	0	0	1	1
6	1	1	0	0	1	1	1	1	3	3
7	1	1	1	0	1	1	1	1	4	3
8	0	1	1	1	1	1	1	0	3	3
9	1	1	1	1	1	1	1	1	4	4
10	1	1	1	1	1	1	0	1	3	4
Tot	6	7	6	5	8	9	5	6	25	27

Where:

N = Novice and
E = Expert.

The novice scorer correctly identified (100 x 25 / 40 =) 62.5% of the lies and the expert scorer correctly identified (100 x 27 / 40 =) 67.5% of the lies.

Appendix D. Plots and discussions of selected SCR responses for the experimental procedure.

Figure D-1. Subject 1, Year.

The third response was identified correctly as the lie by both scorers because it was the last large answer-related response. The large response after the sixth stimulus was associated with a sigh. The abbreviation SCL in the figure titles refers to skin conductance level.

Figure D-2. Subject 2, Year.

The fourth response was identified correctly as the lie by the novice scorer because it was the last large response. The expert scorer selected the second response because it was largest from trough to peak.

Figure D-3. Subject 3, Day.

The third response was identified correctly as the lie by the expert scorer because it was the largest and was followed by a reduction in overall activity. The novice scorer selected the fifth response because it was the last large response.

Figure D-4. Subject 4, Month.

The fourth response was identified correctly as the lie by both scorers because it was the largest and was followed by a reduction in overall activity.

Figure D-5. Subject 5, Card.

The fourth response was not identified correctly as the lie by either scorer. Both scorers selected response six because it was a large response at the end of the series. In retrospect, we saw that the fourth response was the largest.

Figure D-6. Subject 6, Day.

The third response was identified correctly as the lie by both scorers because it was the largest and was followed by a reduction in overall activity.

Figure D-7. Subject 7, Card.

The third response was identified correctly as the lie by both scorers because it was the largest and was followed by a reduction in overall activity.

Figure D-8. Subject 8, Card.

The fourth response was identified correctly as the lie by both scorers because it was the last large response. Even though the response went off scale, it was clear that it contained a much larger area under the curve than the other responses.

Figure D-9. Subject 9, Month.

The third response was identified correctly as the lie by both scorers because it was the largest and was followed by a reduction in overall activity.

Figure D-10. Subject 10, Year.

The fourth response was identified correctly as the lie by both scorers because it was the largest and was followed by a reduction in overall activity.

Figure D-1. S1 Year SCL

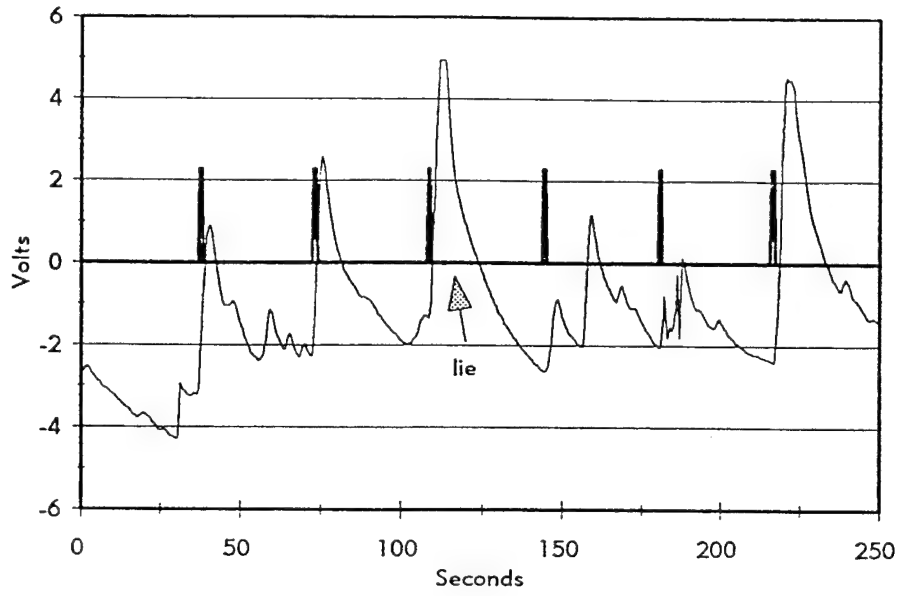

Figure D-2. S2 Year SCL

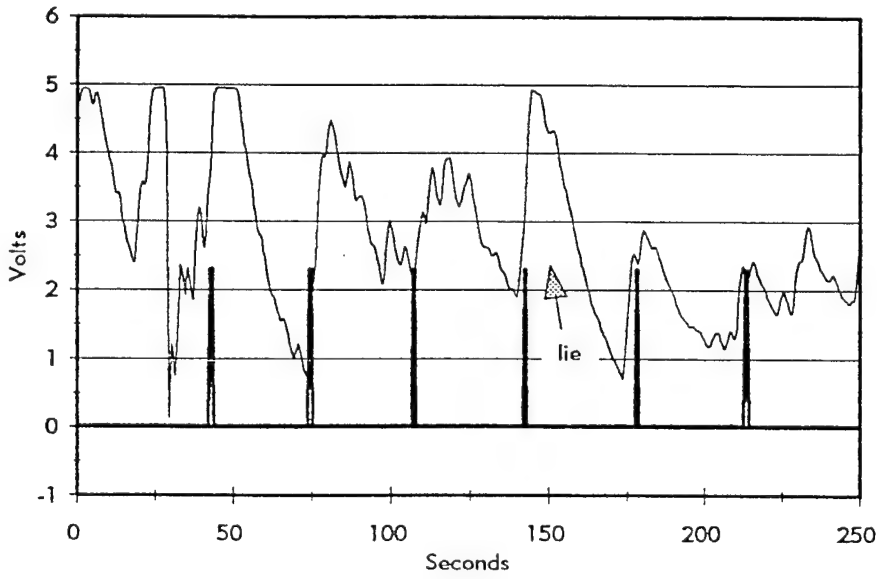

Figure D-3. S3 Day SCL

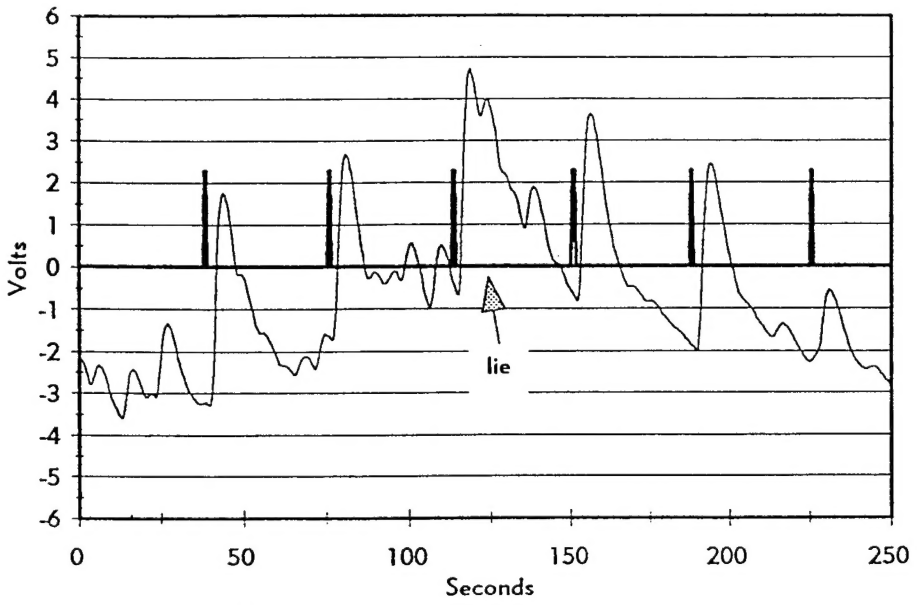

Figure D-4. S4 Month SCL

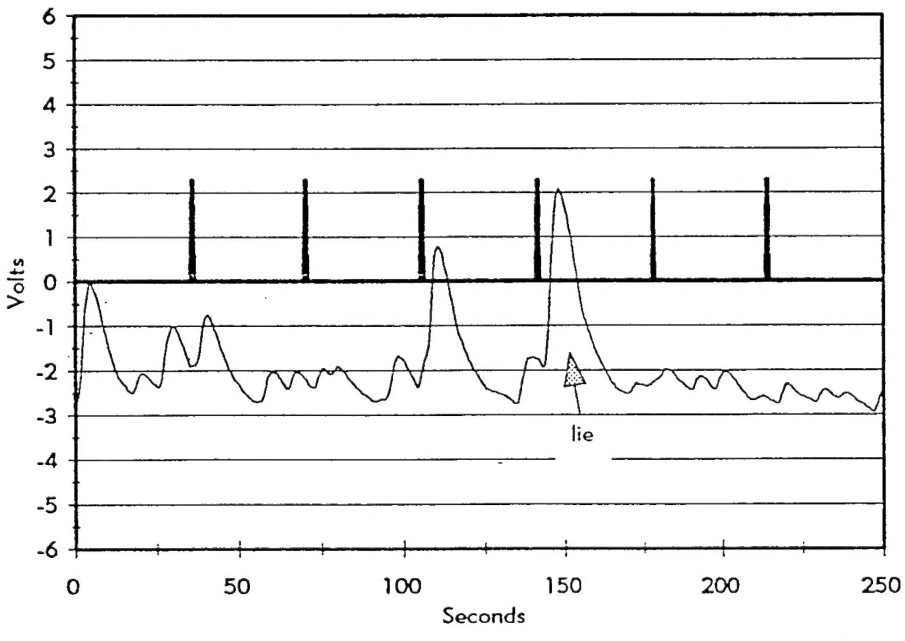

Figure D-5. S5 Day SCL

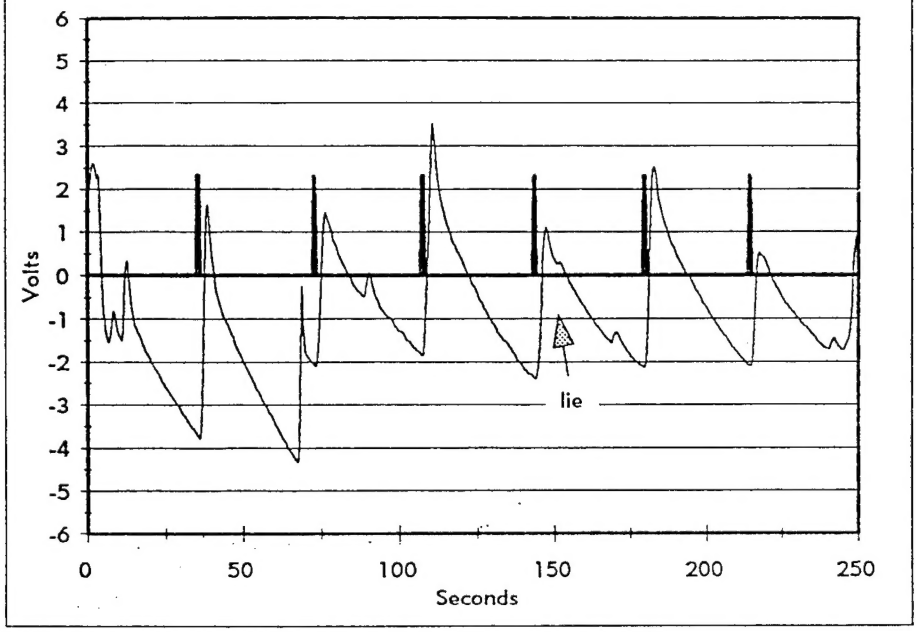

Figure D-6. S6 Day SCL

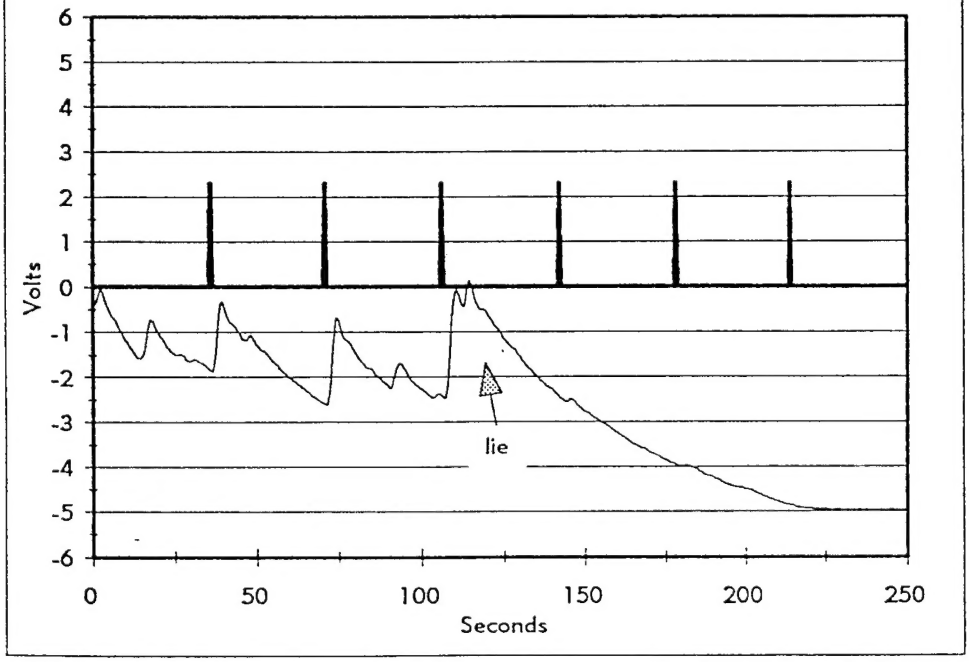

Figure D-7. S7 Cards SCL

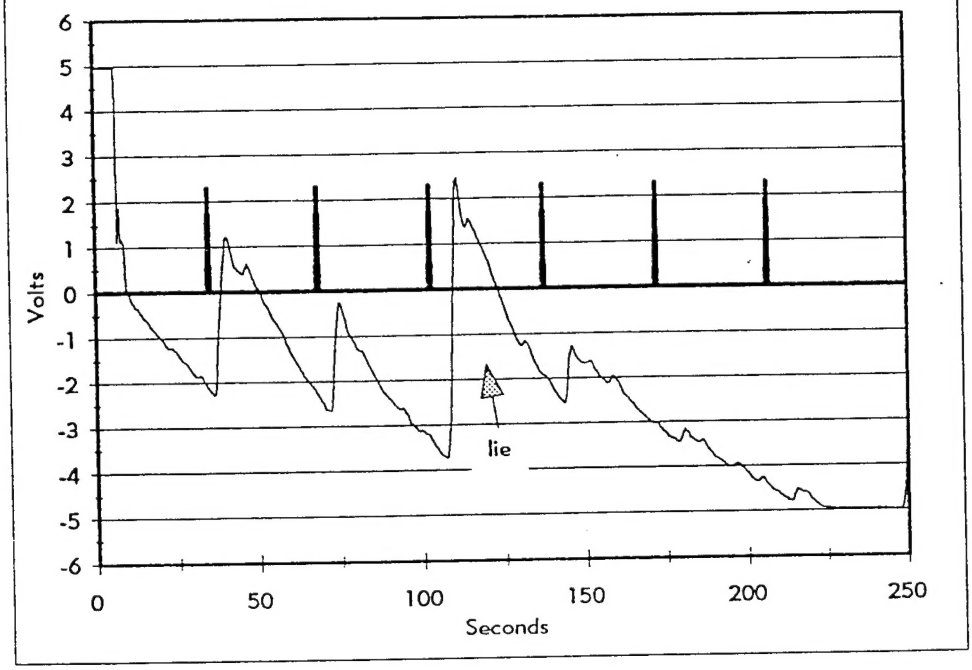

Figure D-8. S8 Cards SCL

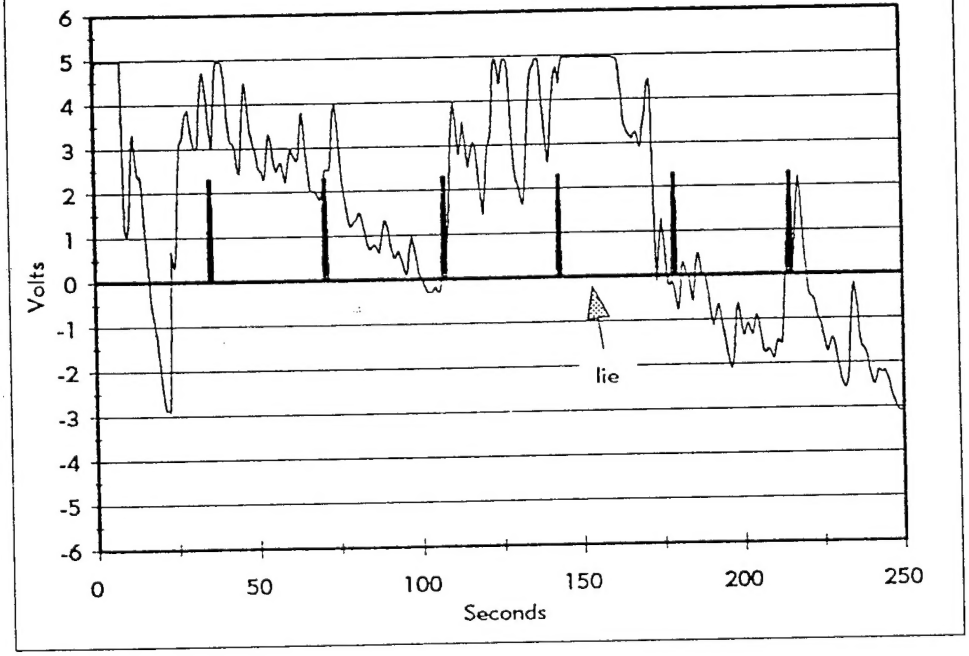

Figure D-9. S9 Month SCL

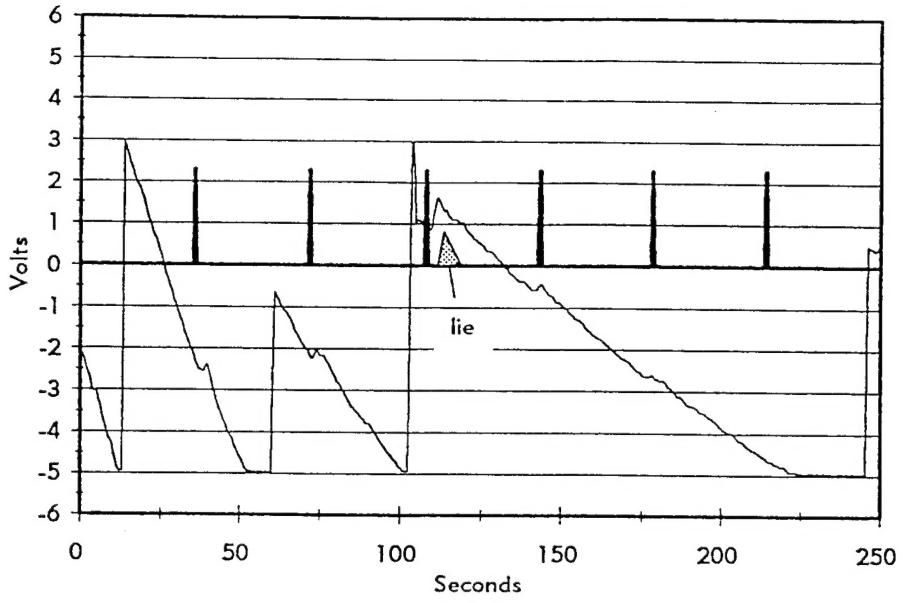

Figure D-10. S10 Year SCL